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ALCOHOL AND LONGEVITY

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ALCOHOL AND LONGEVITY

BY

RAYMOND PEARL

Institute for Biological Research
The Johns Hopkins University

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1926

TO

MY FRIENDS OF

THE SATURDAY NIGHT CLUB

65422

PREFACE

THE investigation which this book principally recounts has occupied my attention intermittently during the past six years. My interest in the general biological effects of alcohol goes back to 1914, when I began the study of its racial influence upon the domestic fowl. The present study of the relation of alcohol to longevity is really a direct outgrowth of that early work. Everyone said that alcohol shortened human life. But alcoholized fowls lived longer than those which got nothing but plain food and water. The torturing curiosity engendered by this apparent discrepancy in the behavior of two forms of life, in some other respects biologically quite similar to each other, only now finds relief as this book is finished.

With the exception of Chapter VIII the material is here published for the first time, in anything like its present form. That chapter follows closely, but with added data, an article published in the Eugenics Review in April, 1924. I am indebted to Mrs. C. S. Hodson, the Secretary of the Eugenics Education Society of London, for permission to use this material here. In Chapter VI the arrangement of the material is in the same order that was used in a review of the literature on alcohol and mortality which I contributed to Starling's well known book, " The Action of Alcohol on Man." But the treatment here is different in many details.

All bibliographic references are put together in a list of *Literature Cited* at the end of the book, where they are consecutively numbered. Citations are indicated in the text by enclosing the ordinal number of the reference in parentheses.

For many different kinds of aid in the preparation of this book I am indebted to my colleague Dr. John Rice Miner. Much of the laborious arithmetic is his, and the dull task of indexing he has most kindly undertaken. For all this I render grateful thanks. Also I am greatly indebted to Miss Blanche F. Pooler, who not only supervised, with great skill and critical acumen, the collection of the original records on which this study is based, but also rendered faithful, timely, and much appreciated aid in the final preparation of the manuscript for the press.

RAYMOND PEARL

June 1, 1926.

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ALCOHOL AND LONGEVITY

"I only thought to make
I knew not what; nor did I undertake
Thereby to please my neighbour; no not I,
I did it mine own self to gratifie."

John Bunyan

CHAPTER I

THE PROBLEM

THE central problem of biology is evolution. While no persons informed as to the evidence and possessed of intelligence sufficient to carry out the simplest processes of logical reasoning, any longer question the fact of organic evolution, the method by which the fact has been and is being accomplished, is not well understood. In the nature of the case the task of finding out what the mechanism of evolutionary change is, must necessarily be an extremely difficult one. The process itself is slow; so slow as to be, in the main, impossible of direct frontal attack by the experimental method. The biologist can, in most instances, study directly and at one time only some single one of the factors which are known to be of importance in the premises, such as, for example, heredity. By these flank attacks, and the general guerrilla warfare of biology in this territory during the last half century, much progress has been made. Great branches, or sub-divisions, of the science have been firmly established. The branch of biology having to do with the phenomena of heredity and variation, which is now called genetics, is a case in point. No longer need we speak with vagueness about the mechanism of the hereditary factor in evolution. Precise knowledge has in large measure replaced speculation and hypothesis.

But brilliant as have been the results of genetic inquiry during the present century, they have exposed and taken, after all, but one flanking outpost of the central citadel. It is the mechanism of evolution as a whole, that we want to know about. The course of biology since the publication of the *Origin of*

Species in 1859 shows with the utmost clearness that many more of these outposts will have to be cleared away before we can uncover the center. Darwin's brilliant frontal attack, the theory of natural selection, we now see to have attained but a partial victory. While natural selection is a process which must always be everywhere in some degree operative, the progress of biological knowledge since Darwin's time has tended to make doubtful two facts: first, as to whether in many particular cases natural selection has in fact operated with a sufficient degree of stringency to produce all of the evolutionary changes which have occurred; and, second, as to whether, in other cases, natural selection can possibly have had anything whatever to do with the initiation or continuation of evolutionary changes.

As an illustration of the first of these points may be taken Dewar's (198) brilliant study, recently published, of the natural checks to the growth of avian population of India. He classifies the destructive agencies which keep down the number of birds into four categories. These are: (1) those agencies operating in a wholly haphazard and indiscriminate manner; (2) those largely indiscriminate in their elimination; (3) those which kill off only the weak and unfit; (4) those which operate mainly on the unfit, but also to some extent selectively. Dewar then examines specific cases of elimination, as observed in the field by himself and other ornithologists, to see how they fall, statistically speaking, in these four categories. His conclusions are (*loc. cit.*, pp. 260–261):

" From the facts that the greatest destruction is to eggs and young birds and that the forces which destroy adult birds for the most part act indiscriminately as opposed to selectively, the inference must be drawn that, speaking generally, the individuals which survive longest in the struggle for existence are the lucky ones, rather than the most fit. It is, of course,

true that, other things being equal, the individuals which are fittest or strongest or best suited to their environment will have the best chance of survival. As a matter of fact other things are rarely equal. The existence of a bird may perhaps not inaptly be compared to a game of cards in which the element of chance is a very important factor. Although it is agreed that *A* is the most skilled of the players taking part in the game, no one, before any particular game, would give long odds on *A* winning, because far more depends on the hands dealt than on the skill of the players. So is it with birds. If we mark down, let us say, four individuals of a species, each living in an area at a distance from the others, it would not be prudent to give odds that the strongest of the four individuals will outlive the other three. The struggle for existence of birds results in the weeding out of the unlucky rather than the less fit.

" In view of the evidence before us it seems difficult to accept the view held by some that all the physical and anatomical features of every species are the result of a process whereby only the fit survive to produce young, which tend to inherit the fitness of their parents. Rather are those characteristics the result of the innate tendency of the majority of individuals of a species to vary in certain definite directions. Natural selection, the survival of the fittest, seems normally to come into action where variations markedly unfavourable to the individual appear.

" Natural selection, then, seems to have played only a very subordinate part in the production of the colouring and the anatomical and physical features of the various species of birds. This, however, does not mean that it has not played its part in evolution. There are conditions in which the force comes actively into play. Natural selection, for instance, has probably helped to determine such features as the number of eggs which compose the normal clutch for each species, the

number of clutches laid in the year, the time of nesting, the situation of the nest, the times of migration and the degree of pugnacity possessed by the average individual of any species."

On the second point, consider the following quotation from an address by Professor William Bateson (199), whose death is such a great loss to biology:

"The survival of the fittest was a plausible account of evolution in broad outline, but failed in application to specific difference. The Darwinian philosophy convinced us that every species must 'make good' in nature if it is to survive, but no one could tell how the differences — often very sharply fixed — which we recognize as specific, do in fact enable the species to make good. The claims of natural selection as the chief factor in the determination of species have consequently been discredited."

It is now generally agreed that, at best, natural selection is only one of a number of factors causally concerned in organic evolution. At present it is impossible to say with any precision or generality what its relative importance as a factor in evolution really is. What is almost universally conceded is that it is a limiting factor, to which all others operative in the premises must conform. That this fact makes it an important element in the case no one would deny. But further than this we cannot safely go at present.

The phenomenon of evolution is necessarily a statistical one. It concerns surviving populations of individuals, all more nearly like each other than like individuals in other and different populations. Quite apart from any and all theories as to the mechanisms involved it is certain that the things which immediately and directly determine the survival of populations are the birth rates and death rates exhibited by the individuals composing them. A population in which the birth rate is constantly higher than the death rate will survive, in the absence

of catastrophic destruction of its component individuals. On the other hand if the death rate is consistently higher than the birth rate it is only a matter of time till the population will disappear. From these considerations the truth of the statement made in the introductory editorial with which the leading journal of quantitative biology, *Biometrika,* was launched nearly a quarter of a century ago, is apparent: "We must ultimately turn to the mathematics of large numbers, to the theory of mass phenomena, to interpret safely our observations. As we cannot follow the growth of nations, without statistics of birth, death, duration of life, marriage and fertility, so it is impossible to follow the changes of any type of life without its vital statistics. The evolutionist has to become in the widest sense of the words a registrar-general for all forms of life."

One of the most important avenues for a flank attack upon the problem of evolution, comparable in its significance to the study of the laws of heredity, is a study of the laws of mortality, in both its direct aspect, and its correlative, duration of life. Duration of life is the most simple and direct measure possible of the survival value of an individual. It tells, in point of fact, just how long he personally survived.

The factors concerned in the determination of the duration of life of the individual include, besides the external, environmental influences acting upon it, all the structural and functional elements in its own make-up. Recent investigations (references 1–17 inclusive) demonstrate that extremely small differences in the structural and functional constitution of the individual, in the case of the fruit-fly *Drosophila,* may have associated with them marked differences in duration of life. This is true of such apparently insignificant things as eye color, the degree of pigmentation of a part of the body, etc. From these facts it follows that in duration of life there exists, ready to the hand of the inquiring biologist, a quantitative index of

the constitutional make-up of the individual. In a sense it may be regarded as an integration of the net biological effects of all the peculiarities of his structural and functional organization. This attribute of the individual, his duration of life, is, then, clearly a character of fundamental biological significance.

With the realization of the theoretical significance for general biology, as well as practical importance for public health, life insurance, etc., of this character duration of life, a major portion of the research energy of the writer's laboratory has been devoted to its study for some years past. The attempt has been made to find out, by experimental and statistical researches, something of the natural laws in accordance with which duration of life is determined. We have studied, on the one hand, the internal factors concerned — the organization, structural and functional, of the individual — which lead to the quasi-inheritance of different degrees of longevity. On the other hand the influence upon duration of life of a variety of external, environmental factors and forces, has been investigated. From this work (in addition to the list of references cited above see also 18–21 inclusive) the broad result which emerges is that while it is the inherited organization or constitution of the individual which basically determines the duration of its life, there are many environmental factors which may modify to a significant degree the inherited potentialities of longevity, and this without acting in any catastrophic manner. One knows, of course, that it is possible to starve an individual to death at any point in its life history by the complete withdrawal of food. Furthermore this can be done with about equal ease to the individual whose inherited constitution gives him the normal expectation of a long life, or to the individual whose constitution is bad or weak, and who in the normal course of events is likely in consequence to die young. But complete starvation is a catastrophic event. It has nothing

to do with the normal influence of diet upon life duration. No one would ever think of attempting to find out what was the effect of food upon duration of life by starving the subjects to death as quickly as possible. Rather one would construct life tables for different groups of individuals, each of which had received a diet throughout life different in some one item, or at most in a few particulars, from those given to the other groups.

So similarly one may give an animal or a man an immediately lethal dose of alcohol. But to do so would teach us only that alcohol is an acute poison when taken in sufficiently large doses. The problem of real interest is to find out what effect alcohol has upon life duration when taken in amounts much smaller than those acutely poisonous, and in fact *apparently* harmless.

Approaching the matter in the way indicated, a problem of great biological interest is presented by the phenomena associated with the influence of ethyl alcohol upon duration of life. It is a substance of marked physiological potency, with which man alone of all the higher animals normally ever comes in contact. It furnishes, in short, an environmental element which normally acts upon only one animal, namely man. This statement is of course not quite literally true. There are some lower animals, notably the common fruit-fly *Drosophila,* which probably ingest a fair amount of alcohol along with their food, in the course of their normal lives. This is true of any animal that normally feeds upon decaying and fermenting fruits and vegetables. But, so far as I know, man is the only vertebrate animal which takes alcohol regularly and normally as a part of its alimentary régime.

Alcohol acts, or may act, upon man in three ways:

a. As a food.
b. As a drug.
c. As a poison.

Alcohol, in moderate amounts, is almost completely oxidized or burned in the body, yielding energy available for use in muscular work, in the maintenance of body temperature, or otherwise. This fact, by definition, makes alcohol a food. It can, within limits, replace equivalent amounts of fats and carbohydrates in the diet, and, within these limits, can spare protein in the same way that fats and carbohydrates can (see for example 22 and 23). In the wine and beer drinking countries of Europe alcohol is extensively used as a normal and useful part of the diet. Dodge and Benedict (24) make the statement (p. 9) that: "Several million people regularly obtain a somewhat larger proportion of their total energy requirement from alcohol than they do from protein." Observation of the lives and habits of the common people of France, Italy and Germany certainly confirms this statement. Carpenter (154) in his most recent study, in which alcohol was administered in the form of rectal enemata, says (p. 184): "The experiments on rectal feeding indicate that the proportion of the total metabolism due to the alcohol injected may be as high as 51 per cent. The utilization of alcohol in rectal feeding therefore plays a prominent rôle in the total metabolism."

In this connection it is interesting to note that Moore and Webster (25) have shown that while methylic alcohol is highly poisonous to simple plant cells (unicellular green algæ), nevertheless if this alcohol is fed to them in sufficiently high dilution, "it can be used as nutrition in absence of carbon dioxide." Marked growth was obtained with this substance as *the sole source of carbon.* Bills (26) has experimented with ethyl, methyl, and other monatomic alcohols, upon the single-celled animal *Paramecium*, and finds that all of these substances postpone the advent of starvation in the cultures, and "may even restore severely starved cultures to their former prosperity." Results apparently the same in purport were got by Camus

(27), who gave four dogs a diet of alcohol and water only, the alcohol being fed either plain or as absinthe. The average duration of life after the beginning of the experiment on this régime was about 37 days. Kochmann (28) also got the same result. He found that when he gave rabbits 3.0 or 3.2 c.c. of 10 per cent alcohol daily, and no food whatever, they lived on the average 21.4 days after starvation was begun, while the control rabbits which similarly were starved but got the same amount of water daily as the others got of alcohol, lived only 17.5 days. This result he explains as due to the protein sparing action of the alcohol.

In a paper recently published, Richter (200) brings forward new evidence of first-rate importance regarding the utilization of alcohol as food. His experiments were made with the white rat, extended over a relatively long period of time, and were carried out with great critical care and accuracy. His technique was remarkable in several respects. In the first place he succeeded, by training the rats early in their lives, in getting them to drink the alcohol, indeed to make their entire fluid intake in the form of solutions of alcohol. By the use of graduated receptacles he measured accurately the daily alcohol consumption, as well as the amount of standardized food ingested. Records were made of the growth and of the activity of these alcoholized rats, and compared with similar records for non-alcoholized controls.

The results attained are stated in the following words (pp. 417–418):

"It was found that the white rat is able to utilize an 8 to 16 per cent solution of alcohol as its steady fluid supply, replacing isodynamic quantities of food without intoxication or habituation effects, but with a definite decrease in spontaneous activity.

"It was shown that the ability of the rat to digest such

large quantities of alcohol without serious effects was due to its high rate of metabolism. The amount of alcohol taken by the rats was 5.1 to 7.8 grams per kilogram body weight. The same amount for a man weighing 70 kilos would mean a daily intake of 357 to 546 grams, or 0.450 to 0.688 liter, which is above what can be taken in without intoxication effects. In Atwater and Benedict's experiments 1.03 grams per kilogram body weight were given, which is one-fifth to one-seventh as much as the rats received. When, however, the difference in energy requirements between rats and man is taken into account, the doses taken by the rats and those given to man in Atwater and Benedict's experiments turn out to be the same. This may be shown in another way by comparing the proportion of the energy of the alcohol to the total energy intake in rats and man. The rats took 1.52 to 2.28 grams; 10.81 to 16.24 calories, or 22.0 to 28.8 per cent of the total energy intake. Atwater and Benedict gave 72 grams per day, that is, 511 calories, or 23.1 per cent of the total energy intake.

"It was found in these experiments, carried on over a long period of time, that rats which took alcohol ate from 16.9 to 35.6 per cent less than controls which drank pure water. In spite of the fact that they ate so much less, they grew just as rapidly and reached the same body weight at maturity as the controls. Under conditions of nearly equal activity the energy of the alcohol, calculated in calories per kilogram body weight, exactly counterbalances the decrease in energy intake. The conclusion was drawn that in the rat alcohol not alone replaces isodynamic quantities of food in maintaining energy balance as was demonstrated in man by Atwater and Benedict, but also that it is used for growth and development."

The final physiological interpretation of these accurate and most significant results, has yet to be made. It is possible that

the action of the alcohol is simply to increase markedly the metabolic utilization of the other constituents of the ration, notably protein, thus lessening the intake requirements of these other constituents in order to maintain a given rate of growth. But, whatever the interpretation, the value of alcohol as a food can no longer be questioned.

Considered as a drug alcohol is essentially purely narcotic in its action. For the most recent authoritative discussion of the physiological and pharmacological effects of alcohol (29) and (30) should be consulted. The notion formerly widely held, that alcohol is a stimulant, has been generally disproved by later and more careful physiological study. Its action on the nervous system is depressant. While the action of alcohol in considerable doses appears always to be depressant upon such physiological functions of higher animals as heart-beat, respiration, etc., there is evidence that it may act as a stimulant to growth. This evidence is in part derived from studies on plants. Puri (155) has recently shown that if ethyl alcohol is added to the nutrient solutions in which barley seedlings are grown, there is indication of a definite stimulating effect in low concentrations. Pearl and Allen (156) have still more recently shown that if canteloupe seeds are soaked for three hours in solutions of ethyl alcohol of different concentrations, germinated, and grown in the dark in distilled water, there is a definite increase in growth rate in the alcoholized seeds as compared with the controls, up to a limiting concentration of the alcohol. Thus the percentage excesses in fresh weight of stems and roots taken together, over the water control seedlings, were for 2 per cent alcohol, 23.0 per cent; for 4 per cent alcohol, 13.6 per cent; for 8 per cent alcohol, 24.3 per cent; for 12 per cent alcohol, 34.8 per cent; and for 16 per cent alcohol, 9.0 per cent. The results of this study are shown graphically in Fig. 1.

Like most other drugs, and indeed like most other chemical compounds in general, alcohol in excessive doses acts as an acute poison. With suitable care in the terminology of the

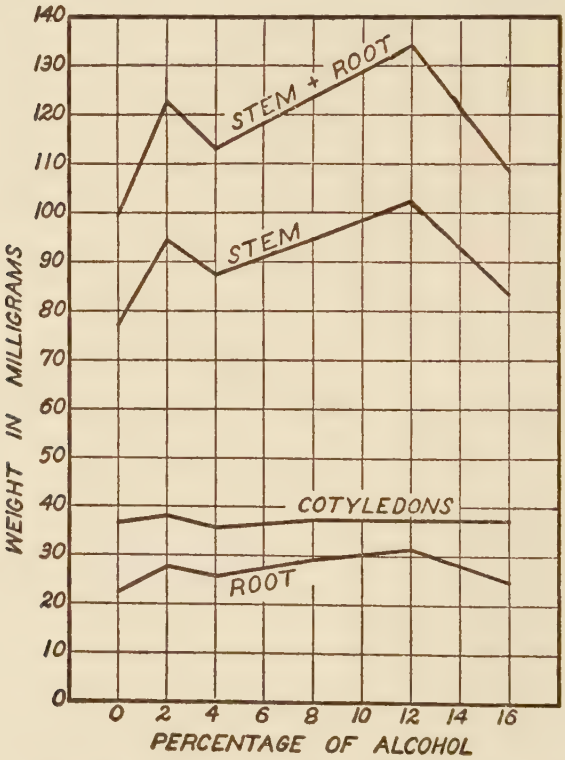

FIG. 1. Mean fresh weight of seedlings of the canteloupe, *Cucumis melo*, after complete growth under conditions of starvation, the seeds having been soaked for 3 hours prior to germination in the indicated solutions of ethyl alcohol (Pearl and Allen).

definition it may also be called a chronic poison, although much of the evidence upon which it has been so regarded will not bear critical examination. In this connection it should be

noted that the careful experimental work of Friedenwald (31) and of Whitney (32) has demonstrated that the toxicity of alcoholic beverages is not simply proportional to their content of ethylic alcohol, and must therefore be due, in part at least, to constituents of these beverages other than the alcohol.

Taken altogether, the known actions of alcohol which have been briefly enumerated suffice to establish its position as a biologically significant element in the environment of man, and therefore of great interest in the study of the problem of evolution. At this point I wish to state, with the greatest possible clearness, that it is in this aspect alone of the alcohol problem, so-called, that I am interested and shall discuss in this volume. That the problem has also important and interesting social and economic aspects is evident. But, as it seems to me, these aspects of the alcohol question are outside the possibility of profitable scientific discussion at the present time. What actually is done by human beings about the social phases of the alcohol problem is primarily and generally determined by emotional and not by rational processes. Interesting as this phenomenon may be to the student of animal (or human) behavior it does not encourage any purely scientific approach to the social problem of alcohol usage. For science can rightly be said to be useful in relation to the conduct of human life only when its results are translated into purposeful action, or at least have some chance of being so translated. I can see no present indication that any scientific result regarding alcohol is likely to have the slightest influence upon legislative or other social action relating to the problem. This seems to me to be certainly and completely true for the United States of America. Yves-Guyot's (33) illuminating and entertaining account of French behavior regarding the matter indicates that the case there differs only quantitatively, not qualitatively. Shand (206), in an introductory discussion

regarding England, indicates the existence of similar conditions in that country.

But, on the other hand, it is interesting and useful for the progress of science to investigate and discuss the purely biological aspects of the effects of alcohol. It is to these that the present work will be wholly confined. I cannot prevent others from making deductions from the data and results here exhibited regarding the social problems presented by alcohol usage; but I can, and do, in the plainest terms disavow all responsibility whatever for any such deductions. I do this not from any desire to avoid responsibility as such, but because, in addition to the reason already set forth, I have an acute realization, on the one hand, of the complexity of the social problem presented by alcohol — of the number of unresolved variables which play important rôles in it — and, on the other hand, of my amateur status in the field of sociology.

The purely biological study of alcohol in relation to evolution falls naturally into two main divisions. These are:

a. The racial or hereditary effect of alcohol.

b. The effect of alcohol upon the duration of life of the individual.

This book deals primarily with the second of these divisions, but one chapter will be devoted to a résumé of the present state of knowledge regarding the racial effects of alcohol.

My own study of alcohol as an environmental agent began more than ten years ago. The original papers dealing with this work are (34) to (37) inclusive. A summary and digest of the results will be found in Chapter VIII of this book. The specific problem to which I addressed myself then was that of experimental modification of the germ cells by this agent. The domestic fowl was the animal experimented upon. Ethyl alcohol was administered daily by the inhalation method. The birds to be alcoholized were placed in closed metal tanks

in which the atmosphere contained alcohol vapor nearly or quite to the point of saturation. For one hour the birds breathed this atmosphere, and in this manner took into the system a relatively large amount of alcohol. The quantity so taken was in the neighborhood of a lethal dose. This was demonstrated by the fact that, under the conditions of these experiments, if the birds were left in the tank for 15 or 20 minutes over the hour they went into a state of coma, and as other experiments showed, would die unless promptly removed to the open air.

This daily treatment with alcohol was kept up with some of the birds for a period of two and a half years. During this time the birds were mated, progeny obtained, and a study made of the racial effects. As already pointed out, this phase of the matter is not the primary concern of the present discussion. But at the same time that the racial investigations were in progress careful observations were made regarding the effect of the treatment upon the physiological economy and life duration of the treated birds themselves, and the results of these observations may profitably be discussed here.

Let us look first at some of the immediate physiological effects of the administration by the inhalation method of ethyl (and methyl, since parallel experiments were run with this) alcohol to fowls, after approximately two years' habituation to its daily intake in the manner described. These observations have to do with respiration rate (respirations per minute) and rectal temperature (in degrees Fahrenheit). They are set forth in Table I. This table gives mean values only. These averages undoubtedly understate the true differences between the groups because they include all the observations, some of which were made on cold days and before the apparatus for maintaining a constant temperature for the evaporation of the alcohol had been perfected. On such days the dosages, and in

consequence the physiological differences, were reduced. By "tanked controls" in the second line is meant birds which were put for one hour in a tank precisely like the alcohol tanks, but in which there was no alcohol or alcohol vapor.

This table shows that:

a. Those birds which, by long-continued daily treatment, extending over periods varying from six months to two years, have become thoroughly habituated to alcohol exhibit a *slower respiration rate* than normal untreated birds of the same breeds, the difference amounting on the average to 4 or 5 respirations per minute. There appears to be no steady constant difference between chronic alcoholists and normals in body temperature.

b. The tanked control birds show no significant change in temperature or respiration as a result of an hour's sojourn in an empty tank.

c. On the contrary the birds which stay one hour daily in a tank containing alcohol vapor exhibit marked and definite changes in their physiological condition at the end of the hour. These changes consist of a *drop in temperature,* amounting on the average to nearly $1\frac{1}{4}^\circ$ F. in the case of methyl treatment, and about half as much in the case of ethyl, and a coincident *increase in respiration rate,* amounting on the average to about ten respirations per minute, or a 50 per cent increase in the ethyl, and to about four and one-half respirations, or a 25 per cent increase in the methyl.

d. These immediate physiological changes are precisely the same in kind and comparable in amount to those which have been shown by Völtz and Baudrexel (38 and 39) to follow the taking of moderate to large doses of ethyl alcohol by drinking, in the case of dogs and men.

The results regarding duration of life unfortunately have to be reported in two parts. Up to the end of fifteen months of

TABLE I

SHOWING THE EFFECT OF THE INHALATION OF ETHYL AND METHYL ALCOHOL VAPOR ON THE TEMPERATURE AND RESPIRATION OF FOWLS HABITUATED TO THESE SUBSTANCES.

Group and Treatment	Rectal Temperature before treatment F.°	Rectal Temperature after treatment F.°	Difference	Respiration per minute before treatment	Respiration per minute after treatment	Difference	Number of birds in group	Number of observations
Mean of normal controls...	107.14	.....	.....	23.77	.....	.....	12	48
Mean of tanked controls ...	107.07	107.13	+ 0.06	22.89	23.67	+ 0.78	4	16
Mean of all ethyls.....	107.07	106.43	− 0.64	18.24	27.86	+ 9.62	7	29
Mean of ethyl females	107.00	106.36	− 0.64	18.24	28.76	+ 10.52	6	25
Mean of all methyls.....	107.14	105.91	− 1.23	18.56	22.50	+ 3.94	4	16
Mean of methyl females ...	107.53	106.45	− 1.08	19.00	23.75	+ 4.75	3	12

the experimental treatment the mortality per cent exposed to risk had been 0 for the alcoholists and 41.0 for their control sisters. In commenting upon this heavy mortality among the controls (more than half of which was due to roup) I said, in 1917 (35, pp. 167–169):

"Roup has existed in endemic form on the Maine Station Poultry plant for many years, as on most other plants where for experimental, or any other purpose, birds are brought in from outside fairly frequently. Ordinarily it gives very little trouble. Occasionally it will break out into an epidemic of greater or less violence, always as a result of relaxation of some routine sanitary or hygienic measure. During the course of this alcohol experiment we have passed through a particularly violent epidemic of the sort mentioned. This fact is reflected in the large proportion of the deaths due to diphtheritic roup or some of its complications. On account of this epidemic the total mortality in the experiment must be regarded as abnormally high. The remarkable thing is that during the fifteen months covered in this report, *i.e.*, to February 1, 1916, not a single one of the treated birds succumbed to this disease, though they were exactly as much exposed to contagion as the controls. This is a surprising result. It seems impossible that it can be due to any real increase in resisting power in the alcoholic birds. A possible explanation is that the daily inhalation treatment acts as a disinfectant of the air passages, and the treated birds do not take the disease because its germs are killed or greatly weakened before they have an opportunity to get an effective foothold. It would be altogether premature to draw any conclusion in regard to the matter until more extended data are at hand. At present I desire merely to put on record the facts now available."

At the time the first report was published (January, 1917) the experiment had gone forward eleven months beyond the

period covered in that report. It was then intended to publish soon a further progress report covering the additional data. But early in 1917 the United States entered the World War, and I was drawn from experimental work into war service. The records were continued by assistants till June, 1917, and for the progeny through the next year. A second misfortune in 1919 led to the destruction of all the records by fire. So the net result is that I can only give an incomplete account, from memory, of what happened in respect of mortality after February 1, 1916, the end of the period covered in the published report. The findings were, however, so striking that, so far as mortality is concerned, the essential facts can be stated with absolute definiteness. The alcoholized birds continued to live under the daily treatment, and finally in June, 1917, when the experiment was closed because of the war, the survivors, which included all but one of those which had started in the experiment with ethyl alcohol, were killed in order that autopsy records of the organs might be obtained. Meantime, all of their control brothers and sisters had long since died.

The general result was perfectly clear. The animals alcoholized daily over a long period far outlived their untreated brothers and sisters. Their mortality rate was lower than the normal at every stage of life. Incidentally the same thing was true of their progeny, both in respect of prenatal and postnatal chick mortality, as the following figures show:

Percentage mortality in first year of progeny of

(a) Alcoholized parents.	Prenatal mortality ...	26.9
	Postnatal mortality ...	10.6
(b) Untreated controls.	Prenatal mortality ...	42.2
	Postnatal mortality ...	36.9

Precisely similar results regarding the effect of alcohol administered by the inhalation method have been obtained by Stockard (40) in his well-known studies on the guinea-pig, as is indicated in the following statement from the memoir cited:

"A number of the guinea-pigs have now been treated with alcohol fumes almost to a state of intoxication six days per week for from five to six years. Few guinea-pigs in captivity live so long a time. There were two males treated for over six years, one of which lived to be more than seven years old. So far as we know this is the longest life reported for a guinea-pig. The treatment was continued with these very old animals, but they were not used for breeding. In no case when the treatment was begun on an animal over three months old could any injurious effects on its general welfare or length of life be discovered." (Page 133.)

"A number of the treated animals have died and many others have been killed at various times during the progress of the experiment. Their organs and tissues have been carefully examined at autopsy and later studied microscopically. All tissues have appeared practically normal, and none of the various well-recognized pathological conditions occurring in human alcoholism have been discovered. Tissues from animals treated as long as three years have been carefully studied, and the heart, stomach, liver, lungs, kidneys, and other organs present no noticeable conditions that might not be found in normal individuals. Alcoholized animals are usually fat, but no fatty accumulation has been noted in the parenchyma of any organ." (Page 134.)

"Our card catalogue contains the record of every death that has occurred among the guinea-pigs since the beginning of the experiments, and we may state in a general way that the

mortality statistics for the treated animals are certainly as good and perhaps slightly better than those of the control." (Page 137.)

Neither Stockard's results nor mine, which agree in showing either a reduced or at least no higher, mortality in alcoholized animals than in those not given alcohol, can be directly transferred to human conditions. One important reason is that the guinea-pig and fowls were given alcohol by the method of inhalation, while man drinks alcohol and does not inhale it. There can be no question that both the immediate and remote physiological effects of alcohol are quite different according as it enters the body through the walls of the stomach or of the lungs. Alcohol taken by the stomach tends to upset the processes of digestion, and if continued over a long period of time in any large amount, tends to derange all the metabolic processes of the body. No such deleterious effects follow upon its inhalation. It is probably much more completely and quickly absorbed when taken by the lungs.

Stockard and Papanicolaou (40) believe that the slowness of absorption from the stomach is the factor of greatest importance in producing the difference in the two methods. They say (pp. 132–133):

"It seems to us, therefore, that the chief difference between inhaling alcohol and drinking it into the stomach is that in the first case the action of the substance on the animal system is of shorter duration, lasting but little longer than the length of the sojourn in the fume tanks — a short acute effect — while alcohol in the stomach is gradually and continuously absorbed for a considerable length of time, so that the animal's tissues are acted upon for hours after receiving the dose. Another very serious phase of the stomach alcohol, aside from the typical intoxication effects, is its tendency to derange the animal's

powers of digestion and thus to cause very injurious results. The inhalation method is accompanied by no such complications."

This statement is not based upon any *ad hoc* physiological or chemical experimentation by the authors. The first point made seems possibly doubtful, in the light of the work of Loewy and von der Heide (41) and of Carpenter and Babcock (42) on certain aspects of the inhalation method of administration of alcohol. Loewy and von der Heide experimented on rats and guinea-pigs and found that considerable amounts of alcohol are taken up by the tissues when it is administered by inhalation. Curiously enough, when administered in this manner, they found methyl alcohol to be less toxic than ethyl. Thus the lethal dose (by inhalation) for rats lies between 3.054 and 5.785 with an average of 4.16 grams per kilo of body weight for ethyl alcohol, while from 8.71 to 12.78 grams per kilo of methyl were required to kill. Guinea-pigs are more resistant. In hens Carpenter and Babcock found up to 5.67 grams per kilo of body weight after inhalation (this was not a lethal dose). They found that the relative concentrations of alcohol in the different tissues after inhalation were as follows:

Blood	100
Heart and lungs	86
Brain	84
Kidneys	79
Alimentary tract	78
Muscle	78
Spleen	75
Liver	72
Skin	56
Fat	11
Whole body	70

These results do not seem to indicate a merely temporary or localized effect of inhalation of alcohol vapor, but rather a widespread diffusion to all the tissues, from which complete elimination is probably slow. Indeed Loewy and von der Heide found that in the case of methyl alcohol given by inhalation there was a definite tendency towards piling up in the tissues (*i.e.*, very slow elimination). Data on the elimination of alcohol through the kidneys will be found in two recent publications (154 and 157).

While the experimental results of Pearl and of Stockard and Papanicolaou on longevity clearly cannot be transferred directly to man because of the different modes of intake of the alcohol, nevertheless they are not without indirect value in the discussion of the human problem. They indicate, so far as they go, that there is no inherently necessary, biologically deleterious effect of ethyl alcohol in moderate doses upon duration of life, however long continued and frequently taken, provided it is administered in a manner which is not immediately and directly harmful physiologically. Or, put in the other way about, the *experimental results prove that ethyl alcohol can be so administered to some living organisms as not to affect harmfully the duration of life.*

These experimental results regarding the effect of alcohol upon longevity were so widely different from the commonly held opinions on the subject as greatly to excite curiosity to investigate the problem with adequate human material. There was in existence one careful study of human data bearing upon the point, and in its results closely accordant with the experimental findings upon lower animals. I refer to Heron's (43) discussion of the morbidity and mortality of extreme inebriates.[1]

[1] This discussion of Heron's work follows, practically *verbatim*, my treatment of the matter in "Alcohol and Mortality" in Starling's *The Action of Alcohol on Man, loc. cit. supra.*

Heron undertook a thorough statistical analysis of data regarding 865 female inebriates, committed to Inebriate Reformatories, between January 1, 1907, and December 31, 1909, the original data having been published by Branthwaite (44) in his annual report. All of the 865 women dealt with were heavy consumers of alcohol, in fact " extreme inebriates " in the literal sense of the words, as indicated by the facts: (1) That they were committed to an Inebriate Reformatory; (2) that they had, on the average, been convicted twenty-four times before for drunkenness, and (3) had a history on admittance of an average of 12.1 years' prior duration of alcoholism.

One section of Heron's memoir (pp. 17–25) is devoted to the discussion of disease and mortality among these extreme inebriates. He shows, first, that these women were distributed as follows, relative to diseased conditions, on the basis of careful physical examination:

No organic disease	519	= 60	per cent
General debility	152	= 18	" "
Heart disease	73	= 8	" "
Syphilis	33	= 4	" "
" Other causes " (including 16 bronchitis, 10 cancer, 6 phthisis)	88	= 10	" "
Total	865	= 100	" "

That 78 per cent of these drunkards, with an average alcoholic history of the whole group of 12.1 years, should exhibit no definite organic disease, when carefully examined by competent medical men, is an astonishing finding, and leads Heron to a more particular examination of the matter. He first attempts a comparison of the general death rate among women committed to Inebriate Reformatories as compared with the

general population of women. Taking the whole experience of the Reformatories which includes 2,767 women under observation 2½ years, he finds actually 39 deaths against 76.7 deaths which would be expected if these women had had the same death rate as the general population. Believing that inebriates come, in general, from particular classes of the population, he attempts a comparison on the assumption that the class from which the female inebriates are chiefly drawn bears the same relationship to the total female population as do general laborers to the total male population. On this basis there would be expected 171 deaths from 2,767 inebriates in 2½ years. Actually, as before mentioned, there were but 39. More precise data from one Reformatory (Langho) gives 13 actual deaths from all causes against 12.5 expected.

By the same method of calculation the mortality from cancer and phthisis was investigated. Assuming the same death rate as in the general population there would be expected in 2,767 women under observation 2½ years 7.9 deaths from cancer and 12.9 from phthisis. Assuming as before that the class from which the female inebriates are drawn bears the same relationship to the total female population as general laborers do to the total male population, the expected deaths from cancer would be 14.9 and from phthisis 34.1. The actual deaths in the experience were 10 from cancer and 6 from phthisis.

One point of detail which Heron notes is that since Inebriate Reformatories were started in England there have been seen in them but two "very doubtful" cases of cirrhosis of the liver. This is interesting in connection with the well-known difficulty of inducing cirrhosis of the liver in experimental chronic alcoholism. Welch (45) says: "Genuine cirrhosis of the liver has not been satisfactorily reproduced by the experimental use of alcohol." Formad (46) found it in only 6 out of 250 autopsies

of confirmed drunkards who had died from the effects of alcoholism. He says (p. 235): "My experience has since taught me that cirrhosis with contraction of the liver is at least as rare an affection in drunkards as it is in 'teetotalers,' and the traditional 'hobnail' or 'gin-drinkers' liver is not diagnostic at all." There is an extensive literature on the relation of alcohol to cirrhosis of the liver. It cannot be reviewed in full or even listed completely in this book, because of limitation of space. References 158 to 197 inclusive will suffice to put the interested reader in contact with the original work of the last twenty-five years in this field.

The general result of Heron's treatment of Branthwaite's data, so far as concerns mortality, is to indicate, on the whole, that the mortality rate among these extreme inebriates is certainly no greater than that prevailing among the classes of the general population from which they are drawn, and perhaps is considerably smaller. What shall we say of this result, which stands in such sharp apparent contradiction to the results of other studies, even the most critical? Do Heron's results represent the real truth of the matter? Or is there something overlooked, which partially or completely invalidates them?

I think there is a factor not taken account of, that may be of importance in the interpretation of the results. It may be put in this way: To what extent are Heron's 865 inebriates the end product of a stringent process of alcoholic natural selection? May they not have survived continued and excessive addiction to be committed to a Reformatory only because of the inborn possession of unusual physical constitution, capable of withstanding without marked harm a deleterious agent which long before eliminated their constitutionally less well-endowed sisters? Heron recognizes this possibility, but his statement (p. 19): "To test this point completely we should require the complete alcoholic histories of a number of women from the

onset of alcoholism, but in the absence of such data it may be said that an average duration of alcoholism of twelve years lends little support to such a view," obviously does not settle the matter. Unfortunately I have no data of my own which are decisive on the point, and I offer the above remarks in no sense as destructive of Heron's results on mortality. I merely wish to point out that until the selection question *is* settled, they cannot be accepted at their face value.

For a number of years the unexpected, but absolutely clean-cut experimental results of Stockard and myself, and the equally unexpected if not quite decisive results of Heron, regarding the influence of alcohol upon longevity, were many times in mind. I had constantly before me the hope that sometime there might be available some reasonably accurate statistical data for man bearing upon the problem in perhaps a more general way than did those with which Heron worked. This hope has now finally been realized. It is the purpose of this book to set forth the results of the analysis of a mass of unique statistical data regarding the influence of alcohol upon longevity. While these records do not reach the highest ideal which one might theoretically set for material with which to investigate the problem, it is nevertheless true that they do give with sufficient accuracy certain important information which has hitherto been lacking.

CHAPTER II

THE MATERIAL

In the experiments with lower animals discussed in the preceding chapter there was available definite knowledge of all the points essential for reaching a critically accurate conclusion as to the influence of alcohol upon duration of life, in the particular forms tested (fowls and guinea-pigs). The experiments start with animals of known age, and of known and uniform genetic constitution. They are kept in a relatively constant environment, and given the same standard food of known composition. They get a definite dosage of alcohol each day throughout their lives. At the same time a group of control animals of the same genetic constitution receive exactly the same treatment in every respect but one, namely that they get no alcohol at all. All the animals, both alcoholized and control, are under observation every day. Their diseases and everything else that may happen to them are known and recorded, at the time of the event. When they die their duration of life can be accurately recorded, and the cause of death, or at least the pathologic status of the body at the time of death, can be determined by performing an autopsy and recording the findings.

All these things together constitute an ideal set of circumstances for studying the problem critically. The records of such experiments, if properly and accurately carried out, furnish just the kind of material needed to answer the question at issue. The case is entirely different when the attempt is made to get a critical scientific answer to the question: What is the

influence of alcohol upon the duration of human life? No records exist which are comparable to those of an experimental inquiry. It is impossible to submit human beings to this kind of experimental study. Even if it could be done, the duration of life of man is so great that the experimenter would almost surely die before he had completed his observations on some of his individual subjects.

We are then forced, in approaching the human problem, to make use of the statistical method of acquiring knowledge, rather than the experimental. This being the case what one would like would be statistics which would follow a considerable number of human beings throughout their lives. Such statistics would record the place and date of birth of each individual, and the circumstances, social, economic and racial, from which he came. They would further tell us of the time of onset, severity and outcome, of all the serious diseases and illnesses which the individual had in his life. In addition to these things there would be recorded the individual's occupation throughout life, his social and economic status and his habits, particularly with reference to the use of alcoholic beverages. The data would be comprehensive enough to include in sufficient numbers persons of all grades of drinking habits, from total abstainers throughout life, on the one hand, to chronic and confirmed inebriates on the other hand. Finally the data would include detailed records of the individual's age at death, and of the cause of his death, as determined by autopsy. From such records it would be possible to construct accurate life tables for persons falling within each defined category in respect of alcohol consumption.

But it is clear that there are no official statistics in existence of the sort described. The registration statistics of births and deaths, in this or any other country, do not give more than the merest fraction of the data outlined in the ideal require-

ments of the preceding paragraph. Nor do census statistics. Nor is there any hope that they ever will. Already the extremely meager and innocuous questions of the census enumerator excite protests against the paternalistic prying of the government into the private affairs of the citizenry. A veritable storm of opposition would follow upon any attempt to collect, officially and generally, statistics about the personal habits of the people.

There is a widespread belief that life insurance companies have in their archives statistical records nearly, if not entirely, fulfilling the ideal requirements of the case set forth above. This belief, however, is based upon a considerable misconception both of the theory and the practical conduct of the life insurance business. The theory of life insurance is essentially this: If an insurance company can bring about the participation in the enterprise of a sufficiently large number of persons who, in respect of their health, constitute a *fair random sample* of the population in general, the company can then figure out with mathematical precision just how much money each person will have to pay down at the time (or distributed over his lifetime) in order to make possible the payment by the company of a stated sum to his heirs at his death. The statistical basis of the enterprise is merely that, *on the average,* a fair random sample of the population will show the same distribution of ages at death as does the general population from which it is drawn. But the distribution of ages at death of the general population is known to a high degree of accuracy from the official mortality statistics of the country as a whole. So then all that the life insurance company theoretically needs to do is to make sure that its policyholders are, in respect of their health at the time of entrance, a fair random sample of the population as a whole. A margin of safety to guard against

unforeseen contingencies like great epidemics, conflagrations, etc., can be further provided in two ways, namely (a) by seeing to it, by medical inspection, that the policyholders at entrance are in some degree in a *better* state of health than the average of the whole population, which result will be accomplished by the simple expedient of refusing policies to all persons who are in a state of ill-health at the time of application, or are otherwise medically bad risks; or (b) by " loading the premiums," which means requiring the policyholder to pay down somewhat more of his money in the form of premiums than the strict mathematical theory of the case says is necessary to enable the company to come out even at the end of any fairly long period of years.

Now, as everybody well knows, life insurance companies do both of these things. They safeguard themselves doubly. It is right that they should. But there is one curious, and from the viewpoint of the present study, important consequence of this solid safeguarding of the financial position and future of the company. It is this. If as a result of medical selection and loading of premiums it is made certain that in the long run the company never can lose money as a result of the mortality of the insured (that is, of the policyholders already in the enterprise) then it is in theory, and to a very large degree in practice, a matter of entire indifference to the company what either the habits or the health of the insured may thereafter become. Nowadays there is nothing except proven fraud which can void a life insurance policy. All companies tend to discriminate, for example, against drinkers who apply for policies. But any person who once has a policy may drink himself to death in a week if he so desires and the company neither can, nor in a broad statistical sense cares to, do anything about it. Indeed for twenty years or more standard life policies have

carried a clause permitting the insured to commit suicide if he wants to, either immediately upon receipt of the policy or after an interval of a year or two.[1]

In consequence of the general non-voidability of an insurance policy once contracted, it results that the companies have no need for and therefore keep no record of the habits or health of the insured, generally speaking, except at the time the policy is taken. It hence follows, from the point of view of the present study, that it is impossible to obtain from the generality of insurance records any exact information as to the alcoholic habits of an individual during his lifetime. The statement of the insured as to his alcoholic habits at the time of application for a policy is of little value for two reasons. In the first place the insured at entrance are apt to be fairly young in average age. Even though they truthfully state what their drinking habits are at that time, in many cases what may be called their *life* habits are not then fully formed. A person at 20 may be a total abstainer and at 35 a heavy drinker. In general, insurance companies will have no records of such facts as these.

In the second place, since it is widely known that insurance companies in some degree discriminate in the issuance of policies against the drinker of alcoholic beverages, there is an incentive to the intending policyholder to understate the facts regarding his alcoholic consumption at the time of application. Of course what the companies are really concerned to do is to exclude the heavy, steady drinker, for the sound reason that he is a poor risk, as will be shown later. The discrimination against the moderate drinker is not, in fact, at all stringent in most companies.

[1] I remember many years ago of an insurance agent urging the personal value of this suicide clause to me, as an inducement to buy a policy from him rather than from a rival company whose contracts did not then contain it!

But from the standpoint of exact scientific inquiry the insurance records can, in the nature of the case, be of but little value. There are a few companies, notably the United Kingdom Temperance and General Provident Institution (46) in England, which offer special financial advantages to total abstainers in the purchase of insurance, who are then required to state annually, if they would continue to enjoy their advantages, that they have remained abstainers. The records of such companies permit an examination of the mortality of the insured divided into two groups, *viz.*, abstainers and drinkers. The latter class is obviously a heterogeneous one, including in unknown proportions (a) truly moderate, and (b) heavy drinkers. It is this fact, as we shall see in later chapters, which is probably chiefly responsible for making the conclusions drawn from such material in some respects misleading. For suppose it to be the fact, for the moment, that the moderate consumption of alcoholic beverages in no wise influences duration of life, while heavy drinking distinctly shortens it, then it follows that if there are included in the non-abstaining section of the insurance experience a number of heavy drinkers (as in fact it would seem that there are always sure to be), the average duration of life in this section will be lowered, in comparison with that in the abstaining section, in direct proportion to the number of such heavy drinkers included. What obviously is wanted is statistical material which will, first of all, record the alcoholic habits throughout life, and also enable a critical separation of moderate and heavy drinkers to be made.

In order to get satisfactory data for the study of this problem it is plainly necessary to collect them *ad hoc*. This we have done. The material so gathered forms the basis of the present book.

The general features and history of the material (19 and

49) have been described elsewhere. I shall follow that account here, supplementing it with additional details where necessary. The material was extracted from the Family History Records of the Institute for Biological Research of the Johns Hopkins University. These Family History Records consist fundamentally of elaborate and detailed original pedigrees, collected by a staff of field workers working under my direction, and under the immediate supervision of Miss Blanche F. Pooler. The workers were especially trained here for this investigation. In most instances they had in addition previous experience in some form of social work, as well as general training in biology.

Some six years ago, at the request of the National Tuberculosis Association, and with its financial support at the outstart, and that of the Russell Sage Foundation and the Commonwealth Fund later, I began what was intended to be a comprehensive investigation of the factors involved in the etiology of tuberculosis, with special reference to the genetic elements in the case. The plan of the work, in outline, was as follows: It was proposed to collect a large number of much more detailed and elaborate family histories of tuberculous persons than any that had hitherto been compiled, doing this by means of field or social workers. These field workers visited the families of individuals whom it was desired to investigate, and got by personal interview the information to be set down in the histories. Proceeding in the matter in an entirely objective way and without preconceptions, it was felt to be essential to get the most elaborate and critically exact records possible about the environmental situation, the habits of life, the health history, the racial stock, anthropological characteristics, exposure to tuberculosis, etc., of all the members of the family group.

The histories were, from the first, divided into two groups. In one set the history starts with an individual, the *propositus*, who is known to have clinically manifest tuberculosis. In the

other set each history starts with a *propositus* who is definitely known *not* to have tuberculosis in any clinically discoverable form. The initial individuals for these two sets of histories were obtained in the following ways. The tuberculous individuals were taken at random (except for race stock) from among those persons who were registered with the Baltimore City Health Department as having active tuberculosis, under the law which makes this a reportable disease; or from those persons registered with Phipps Tuberculosis Clinic of the Johns Hopkins Hospital. The non-tuberculous individuals were taken at random (except for race stock) either from among those persons who had, for some trivial offense (such as, for example, playing baseball in a vacant lot, etc.), been before the Juvenile Court and were known not to be tuberculous; or from patients registered at the General Dispensary of the Johns Hopkins Hospital, and known not to be tuberculous.

Starting from the *propositus* the procedure was the same in both sets of histories. Through the work of the field workers the family history of the individual was traced, both in respect of the ancestry and their collaterals, and in respect of descendants and their collaterals. The histories record in every respect identically the same kind of facts in the two groups. The only difference is that in the one case they start with an individual known to be tuberculous, and in the other case they start with an individual known not to be tuberculous. The same questions are asked and the same facts recorded for all the individuals in the family tree in both sets.

Every critical safeguard of the accuracy of the ultimate individual records that we were able to think of was thrown around this work. No material fact was entered in a history finally until it had been corroborated by the independent testimony of at least two persons acquainted with the individuals in ques-

tion. The material has all been taken from one socially and economically homogeneous group of the population of the city of Baltimore and Maryland; namely, what might inclusively be called workingmen's families. By the employment of field workers speaking a variety of foreign languages it was possible to get representation in the histories of the different foreign race stocks in about the proportion that they are represented in the total population of the city of Baltimore. In this way there was no racial discrimination in the study. The only exception to this is that the negro was not included, owing to the difficulty — indeed impossibility — of getting reliable genealogical information about negro families.

The elaborate records which were taken regarding the environmental surroundings of the persons in the histories demonstrates that they may be regarded as forming a homogeneous group in this respect.

Particular attention in the study was paid to the habits of the individual relative to alcohol and tobacco. This was done because of the possible significance of differences in these habits as a factor in the etiology of clinical tuberculosis. The workers were instructed to find out, in as great detail as possible, the drinking habits of the persons studied. In introducing this subject when talking with members of the family, the workers explained and defended their interest in the matter on the ground that alcohol might be a significant element in the causation of tuberculosis, and that it was thought extremely important to find out whether this was so or not. Neither the field workers nor the persons interviewed had the slightest idea that the material so collected was ever to be used to study the problem of the influence of alcohol upon life duration. Because of the obviously reasonable nature of the request for information on this point in connection with the study of tuberculosis, it was usually given frankly and with-

out hesitation, and without reservation or bias, so far as can be determined by any checks it has been possible to devise.

The records so obtained have these significant characteristics:

1. For each individual included in the statistics there is a definite record, varying, of course, in different individual cases as to the amount of detail included, regarding the kind and amount of alcoholic beverages used, and the frequency or regularity of such usage. If a person had the habit to take four or five drinks of whiskey per day the fact is so recorded; and similarly if a person of the Jewish faith took only a small amount of wine weekly in connection with the observance of the ritual of that religion, this fact is definitely recorded.

2. The habits as to alcohol usage at different periods through life are definitely recorded. In the case of individuals still living at the time when the records were taken, the information under this and the preceding paragraph was usually obtained by the field workers from the individual concerned and from some other member of his or her immediate family. In case the individual could not be reached by the field worker, because of recent migration, the information was derived from two close relatives thoroughly acquainted with the life and habits of the person in question. In case of persons who had died before the inquiry was made, the information was obtained from at least two of the close relatives, who again were entirely familiar with the life of the individual in question.

These two items of information comprised in paragraphs 1 and 2 above sharply differentiate the present material from anything available for the study of this problem in the records of insurance companies, or indeed anywhere else, so far as is known to the writer. They would seem to furnish the basis for a more precise and detailed classification of the individuals in the experience, regarding their alcoholic habits, than anything

hitherto available. It is primarily due to this circumstance, in my opinion, that the results obtained are in certain respects different from those which have hitherto been obtained from a study of insurance records.

Naturally such records cannot, in every case, have either the comprehensiveness as to detail, or the precise accuracy, of experimental laboratory records. We have tried, however, to make them as accurate and detailed as is humanly possible. All general statements such as " moderate," " heavy," etc., we have endeavored to quantify and complete, by getting the individual's own statement as to the amount meant by the general term used in that particular case, and the kind of alcoholic beverage or beverages generally used. With all such efforts it was impossible to get definite information on this point for many of the individuals included in the histories. The result is that the numbers whose records finally passed our critical standards are by no means as large as we should like them to be. On the other hand, it is probable that for the numbers we do have, the information as to drinking habits is more accurate and comprehensive than any elsewhere available.

3. For each individual in the statistics, the age is definitely known. In the case of individuals still living at the time the record was taken, the recorded age is their living age at that time. For persons who died before the record was taken, the recorded age is that at death. Also, of course, the sex of each individual is recorded.

4. For each individual included in the statistics the cause of death was recorded, if the person had died and it was possible to obtain this information. In many cases the cause of death given in these statistics is that recorded on the original death certificate, from which our entry was copied. Where the person had died outside of Baltimore, or for some other reason it was not possible to obtain access to the original death

certificate, the cause of death recorded is that stated to have been the fact by some close relative. In these cases the statement must, of course, be accepted with great caution. It should be said, however, that a comparison of family statements as to the cause of death with those made on the official death certificates leads one to conclude that the former have an unexpectedly high reliability. Besides the death record great pains were taken to find out whether each individual, including both the living and the dead, had at the time of the record, or ever had had, any one of the following diseases:

Tuberculosis,

Heart disease,

Pneumonia,

Bright's disease, or any other disease of the kidneys recognized as such,

Cancer,

Insanity.

5. Records were made of the general social status and economic circumstances of the families to which the persons included in the records belong. The extent of this information varies a good deal in the records. In some there are pages of details as to the habits and circumstances of the family. In other cases only the barest details on a few simple points could be elicited. The coöperative spirit of the people involved is the determining factor on this point.

6. The racial origin of the individuals was recorded in as precise and detailed a manner as the circumstances of the case permitted. The population of a fairly old American city like Baltimore, however, must obviously be to a considerable degree racially mixed, and the best one can do is to make a record of the different elements which have gone into the constitution of the individual during a few generations past. In a comparatively small number of cases the families were genuinely

ignorant of even their broad racial origins, having been so long in this country. The only conscious selection anywhere practised in the collection of the material (except of course as to whether the *propositus* had or did not have tuberculosis) was in respect of race stocks. Families of different race stocks (excluding entirely the negro) were chosen for study, in roughly the proportion that these stocks were represented in the population of Baltimore.

Having considered the method of collection of the material, we may next examine some of its general characteristics, particularly with reference to racial composition, economic status and magnitude.

The absolute numbers of persons belonging to the several race stocks included are shown in Table II. This table exhibits as well the number of males and of females belonging in each of three broad categories in respect of alcohol consumption. The definitions of these categories will be discussed in detail in the next chapter. Here it will suffice to say that, in the sense here used, an " abstainer " is one who takes no alcohol; a " moderate " is one who is in the habit of drinking in moderate amount either occasionally or steadily, but never to the extent of getting drunk; a " heavy " is a person in the habit of getting drunk, either occasionally or frequently.

In Table II, the meaning of the rubrics as to racial origin is as follows:

I. *Scandinavian.* Includes only persons of purely Swedish, Norwegian or Danish origin, and mixtures in which these stocks predominate.

II. *German only.* Includes only persons of purely Germanic origin.

III. *German and British.* Includes persons from families in which Germanic and British (as defined below) stocks were, so far as could be determined, about equally represented.

TABLE II

TOTAL EXPERIENCE, BY RACIAL ORIGIN, ALCOHOL CONSUMPTION, AND SEX.

Racial Origin	Abstainer		Moderate		Heavy		Total		Grand Total
	Male	Female	Male	Female	Male	Female	Male	Female	
Scandinavian	5	10	9	4	4	3	18	17	35
German only	246	342	316	136	236	12	798	490	1288
German and British equally	103	156	119	41	86	7	308	204	512
German and other than British equally	58	110	73	29	66	8	197	147	344
All other combinations involving German, and not proponderantly British	19	41	36	1	17	2	72	44	116
Slavonic only	25	41	199	178	62	6	286	225	511
Hebrew, including all combinations of Hebrew	67	103	109	65	16	0	192	168	360
English only	82	118	43	8	56	7	181	133	314
Irish only	65	130	62	25	56	6	183	161	344
Scotch or Welsh only	12	22	9	1	8	0	29	23	52
Purely British combinations only	64	96	65	30	37	3	166	129	295
Preponderantly British combinations	73	116	97	21	94	6	264	143	407
Italian	26	39	99	78	32	3	157	120	277
Other Mediterranean, and combinations involving Mediterranean, not elsewhere included	28	42	23	6	24	2	75	50	125
Old American	20	44	18	4	21	0	59	48	107
All other	33	42	36	18	30	2	99	62	161
Totals	926	1452	1313	645	845	67	3084	2164	5248

These were German-English, German-Irish, and German-Scotch.

IV. *German and other than British.* Includes the following mixtures of race stocks: German-French, German-Old American, German-Spanish, Russian-German, German-Italian, German-Bohemian.

V. *All other combinations involving German, and not preponderantly British.* Includes the following mixtures: German-Irish-French, German-Dutch-English-Old American, German-Dutch-English-Scotch-French, Scotch-Cuban-German-Irish, German-English-French, German-English-Italian, German-Polish-Irish, Italian-German-Irish, German-Dutch-English-Irish.

VI. *Slavonic.* Including only persons of either Russian, or Slovak, or Lithuanian, or Polish, or Magyar, or Bohemian origin.

VII. *Hebrew.* Includes, first, persons of purely Hebrew origin, including Russian, German, Polish, Rumanian and Austrian Hebrews. In addition this class includes the following combinations involving Hebrew as one element; German-Hebrew-Old American, German-French-Hebrew-Old American, Russian and German Hebrew, Austrian and Russian Hebrew, German-Hebrew-English-Old American.

VIII. *English only.* Includes only persons of purely English ancestry.

IX. *Irish only.* Includes only persons of purely Irish ancestry.

X. *Scotch or Welsh only.* Includes only persons whose ancestry was purely Scotch or purely Welsh.

XI. *Purely British combinations only.* Includes the following mixtures: English-Irish, Scotch-Irish, English-Scotch, English-Irish-Scotch, English-Irish-Scotch-Welsh.

XII. *Preponderantly British combinations.* Includes the

following racial mixtures: Irish-Scotch-French, German-Irish-English-Scotch, English-Irish-French, English-Scotch-Old American, German-English-Irish, German-Irish-Scotch, German-English-Scotch, Dutch-English-Irish-Scotch, Dutch-English-Irish, German-English-Irish-Scotch-Old American.

XIII. *Italian.* Includes only persons of purely Italian ancestry.

XIV. *Other Mediterranean.* Includes the following groups: Irish-Italian, English-Scotch-French-Old American, French, French-English, Scotch-French, French-Irish, Dutch-French, Dalmatian-French-Belgian.

XV. *Old American.* Includes persons born in the United States about whose racial origin nothing could be learned except that their ancestors had been long in this country. In using the designation "Old American" for this group I follow the example of Hrdlička (266).

XVI. *All others.* Includes the following stocks and mixtures: Irish-Old American, Dutch-Scotch, Russian-Polish, Dutch-English, Polish-Bohemian, Dutch-Welsh, German-Irish-Scotch-Old American, Bohemian-Irish, Dutch-Irish, Magyar-Bohemian, Polish-Irish.

In order that a clearer grasp of the meaning of the data of Table II may be had, Table III is next presented. In Table III the absolute frequencies of Table II are reduced to percentage form, to show the proportionate number of persons of each race and sex group in each of the three broad categories of alcohol consumption.

This table is to be read in the following manner: Of the 798 male Germans in the experience 30.8 per cent were total abstainers, 39.6 per cent were moderate drinkers, and 29.6 per cent were heavy drinkers. Similarly of the 490 German females 69.8 per cent were abstainers, 27.8 were moderate drinkers, and 2.4 per cent were heavy drinkers.

TABLE III

TOTAL EXPERIENCE, BY RACIAL ORIGIN, ALCOHOL CONSUMPTION, AND SEX. PERCENTAGES OF ABSTAINERS, MODERATE, AND HEAVY DRINKERS IN EACH SEX.

Racial Origin	Abstainers		Moderate		Heavy	
	Male %	Female %	Male %	Female %	Male %	Female %
I. Scandinavian only	27.8	58.8	50.0	23.5	22.2	17.6
II. German only	30.8	69.8	39.6	27.8	29.6	2.4
III. German and British equally	33 4	76.5	38.6	20 1	27.9	3.4
IV. German and other than British equally	29.4	74.8	37.1	19.7	33.5	5.4
V. All other combinations involving German, and not preponderantly British	26.4	93.2	50.0	2.3	23.6	4.5
VI. Slavonic only	8.7	18.2	69.6	79.1	21.7	2.7
VII. Hebrew, including all combinations of Hebrew	34.9	61.3	56.8	38.7	8.3	0
VIII. English only	45.3	88.7	23.8	6.0	30.9	5.3
IX. Irish only	35.5	80.7	33.9	15.5	30.6	3.7
X. Scotch or Welsh only	41.4	95.7	31.0	4.3	27.6	0
XI. Purely British combinations only	38.6	74.4	39.2	23.3	22.3	2.3
XII. Preponderantly British combinations	27.7	81.1	36.7	14.7	35.6	4.2
XIII. Italian	16.6	32.5	63.1	65.0	20.4	2.5
XIV. Other Mediterranean, and combinations involving Mediterranean, not elsewhere included	37.3	84 0	30 7	12.0	32.0	4.0
XV. Old American	33.9	91.7	30.5	8.3	35.6	0
XVI. All other	33.3	67.7	36.4	29.0	30.3	3.2
Totals	30.0	67.1	42.6	29.8	27.4	3.1

Taking the table as a whole it is seen that the percentage of male abstainers varied from 8.7, in the case of racial group VI (Slavonic), to 45.3 in the VIII (English), with an average for the whole experience of 30.0 per cent. The Slavonic group is a small one (286 males in total). They obviously retain in this country their European drinking habits. The same is true of Italian males (Group XIII). Leaving out these two groups it is seen that there is relatively little variation among the racial groups in the percentage of male abstainers.

The extreme groups in the proportion of males in the moderate class are the same as for the abstainer class, but with reversed position. The English (Group VIII) has the lowest percentage of moderate drinkers, 23.8; while the Slavonic group (VI) has the highest percentage, 69.6. The Italians (XIII) are nearly as high. The average percentage of moderate drinking males for the whole group is 42.6.

The lowest percentage of heavy drinking males is found in the Hebrews (Group VII), with a figure of 8.3. The highest percentage of heavy consumers is 35.6, found in both Group XII (Preponderantly British combinations) and Group XV (Old American). Of all males in the experience 27.4 per cent were heavy drinkers.

Taking the data as a whole there is a greater approach to uniformity amongst the several racial groups, in respect of proportionate distribution of males in the three categories of alcohol consumption, than perhaps would have been expected. With the few exceptions that have been indicated, the tendency plainly is for the percentage values for males to cluster fairly closely about the mean values. So far as the males are concerned there certainly is no reason why the experience from different racial groups should not be put together for statistical treatment. No significant error will be made by so doing. The few widely divergent groups are numerically not large.

Turning to the females there is found, as would be expected for several reasons, a greater degree of diversity between the different racial groups in respect of drinking habits than among the males. To begin with the absolute numbers involved are smaller, a fact which in itself would tend to make the statistical ratios less stable. Quite apart from this factor, however, it is probable that such influences as religious beliefs, home conditions, and other social factors, tend to wider differentiations in respect of drinking habits in females than in males. The general economic and social status (in Baltimore) of an Italian family and an American family of purely English origin may be exactly the same so far as can be externally observed, yet the drinking habits of the women of the two families be totally different. The English family may be strict Methodists and behave as such, while the Catholic faith of the Italians sits as comfortably as an old coat upon their shoulders, without the slightest suggestion that any member of the family, male or female, should forgo the moderate drinking of wine with meals.

Kirby (67) notes, in a discussion of a series of 1762 admissions to the Manhattan State Hospital for insane patients from New York City, that alcoholic insanity is much more frequent among the Irish than among Jewish or Italian patients. Since alcoholic insanity has as its regular precursor heavy drinking, his data are of some interest in the present connection. He found in 336 Irish admitted that 20 per cent had alcoholic insanity (what this really means is probably no more than that of 336 insane Irish 20 per cent were admittedly or notoriously heavy drinkers). Among 455 insane Hebrews only 0.6 per cent were alcoholic, and in 123 Italian patients 5 per cent were alcoholic. These figures disregard sex. Similarly disregarding sex it is seen from Table II that of 344 Irish in the present experience 62, or 18 per cent, are heavy drinkers. Of the 277 Italians in the present experience 35, or just under 13 per cent,

TABLE IV

TOTAL EXPERIENCE, BY BROAD CATEGORIES OF RACIAL ORIGIN, ALCOHOL CONSUMPTION, AND SEX

Racial Origin	Abstainer		Moderate		Heavy		Totals		Grand Total
	Male	Female	Male	Female	Male	Female	Male	Female	
Germanic	323	493	425	166	319	22	1067	681	1748
British	296	482	276	85	251	22	823	589	1412
Germanic and British equally	103	156	119	41	86	7	308	204	512
All other	204	321	493	353	189	16	886	690	1576
Totals	926	1452	1313	645	845	67	3084	2164	5248

are heavy drinkers. Curiously enough Kirby notes that in his group not a single Jewish woman had alcoholic insanity, and in the 360 Jews in the present experience no woman is a heavy drinker. While obviously a comparison between a sample composed of insane persons and a sample of the general working class population cannot be pushed far, it is interesting to see how racial habits in respect of alcohol roughly maintain themselves under various circumstances.

Having exhibited the racial origin of the material in detail, it will be desirable to consider it in broader categories in respect of race, to the end that a more general view of this aspect of the matter may be had. Table IV (on page 49) gives the figures of Table II rearranged.

It is at once apparent from Table IV that a great majority of the persons included in the experience are of either Germanic or British origin, or have combined in them these two broad stocks. That this would be bound to be the case in any large sample of the working class population of Baltimore must be evident to anyone acquainted with the history and present condition of the city. This fact is brought out in relative figures in Table V.

TABLE V

PERCENTAGES OF BROAD RACIAL GROUPS AMONG ABSTAINERS, MODERATE, AND HEAVY DRINKERS

Racial Origin	Abstainers		Moderate		Heavy		Totals	
	Male %	Female %	Male %	Female %	Male %	Female %	Male %	Female %
Germanic	34.9	34.0	32.4	25.7	37.8	32.8	34.6	31.5
British	32.0	33.2	21.0	13.2	29.7	32.8	26.7	27.2
Germanic and British equally	11.1	10.7	9.1	6.4	10.2	10.4	10.0	9.4
All other	22.0	22.1	37.5	54.7	22 4	23.9	28.7	31.9
Totals	100	100	100	100	100	100	100	100

It is seen that of the male abstainers 78 per cent are of Germanic or British origin, or both. For the female abstainers the figure is almost exactly the same (77.9). Nearly the same percentages (77.7 and 76.0) hold for male and female heavy drinkers. In the case of moderate drinkers, the proportion of Germanic and British persons in our sample is smaller. Only 62.5 per cent of the moderate drinking males, and 45.3 per cent of the moderate drinking females, belong to this Germanic-British stock. The Slavonic, Hebrew and Italian groups in the "All other" category of Tables IV and V contribute many moderate drinkers.

Table VI, which is derived from Table IV, presents the differences in drinking habits of the broad racial groups in a clear manner.

TABLE VI

PERCENTAGES OF ABSTAINERS, MODERATE, AND HEAVY DRINKERS IN EACH SEX

Racial Origin	Abstainers		Moderate		Heavy		Totals	
	Male %	Female %	Male %	Female %	Male %	Female %	Male %	Female %
Germanic	30.3	72.4	39.8	24.4	29.9	3.2	100	100
British	36.0	81.8	33.5	14.4	30.5	3.7	100	100
Germanic and British equally	33.4	76.5	38.6	20.1	27.9	3.4	100	100
All other	23.0	46.5	55.6	51.2	21.3	2.3	100	100
Totals	30.0	67.1	42.6	29.8	27.4	3.1	100	100

The data of Table VI are shown graphically in Figs. 2 and 3.

The first fact which these data emphasize strongly is the sex difference in drinking habits. The total abstainer and heavy groups have each roughly about the same proportion as the other of the total population of males. This relation holds approximately for each of the four broad racial groups. The

proportion of male moderates is somewhat, though not greatly, larger than the proportion of either abstainers or heavy drinkers, in the Germanic and British groups. In the "All other"

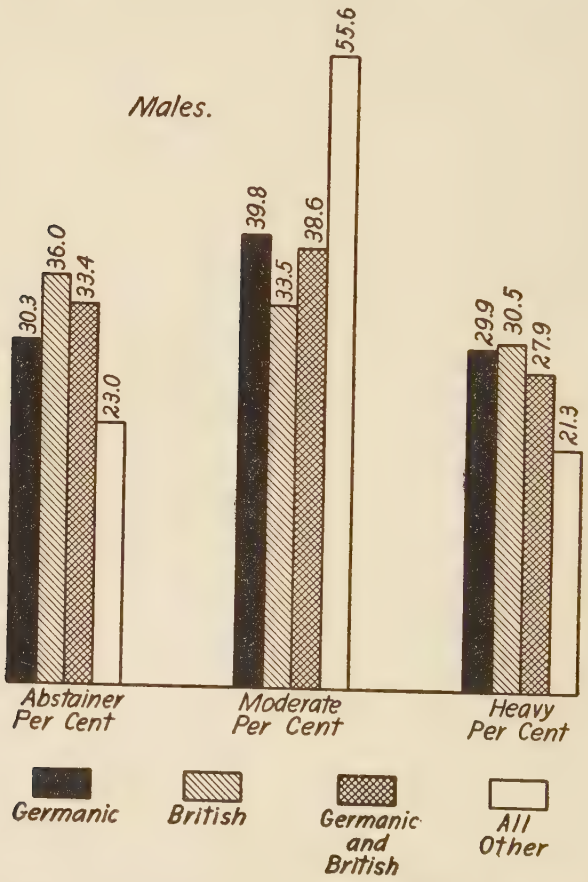

FIG. 2. The distribution of four broad racial groups in respect of drinking habits. Males.

group the percentage of moderate males is more than twice as great as that of either abstaining or heavy drinking males.

FIG. 3. The distribution of four broad racial groups in respect of drinking habits. Females.

The female picture is quite different. In the Germanic and British stocks the great majority (roughly three-quarters) of the females are abstainers. About one-fifth are moderate drinkers, and only 3 to 4 per cent are heavy drinkers. In the "All other" group of females about the same proportion (2.3 per cent) are heavy drinkers, but the remaining females in this group are nearly evenly divided between the total abstainers and moderates.

Data at all comparable to these regarding drinking habits in a sample of an American population are not numerous. Those of Reed (50) are interesting. In 200 consecutive cases of mental and nervous diseases, coming to him in his private practice as a neurologist, 16 were children. The remaining 184 adults were distributed in respect of alcoholic habits as shown in Table VII.

TABLE VII

REED'S DATA AS TO ALCOHOLIC HABITS

Drinking Category	Males		Females	
	Absolute	Per cent	Absolute	Per cent
Abstainers	22	26.8	79	77.4
Moderate	47	57.3	21	20.6
Excessive	13	15.9	2	2.0
Totals	82	100.0	102	100.0

There is obviously a general similarity between the percentages of Table VII and those of Table VI. The males in Reed's series show lower percentages of both abstainers and heavy drinkers than does the present material, and a correspondingly higher percentage of moderates. Probably he classed some persons as moderates who would in the present work be placed with the heavies, for reasons to be discussed in the next chap-

ter. In the case of the females his percentage of abstainers is higher than that shown by the present material, and that of drinkers correspondingly lower. In considerable part the differences between the two sets of percentages are probably to be explained on the basis that this material undoubtedly is drawn from higher social strata, on the average, than ours. The differences are of just the sort that would be expected if this were true. Reed himself believes that the proportion of total abstainers in his material is larger than would be found in the general population, and goes on to conclude that there is as much justification for assuming that total abstinence predisposes to nervous and mental diseases as that moderate drinking does.

There are some interesting data in the literature on the percentage of heavy drinking females found in different groups of the population, which may be considered here. From Tables IV and VI it is seen that, in the present material, out of 2164 females 67, or 3.1 per cent, fell in the heavy drinking class. Furthermore it is to be noted from Table IV that in the present material there are, all told, 912 persons in the heavy drinking category, of which 67, or 7.35 per cent, were females. Maupaté and Nollens (64) report from the Asile of Bailleul, which receives all the indigent insane women of the Nord, in 1085 insane women 84, or 7.84 per cent, who were inebriates. Hultgen (51) found among heavy alcoholics (delirium tremens, alcoholic psychoses and similar cases) admitted to the Cook County Hospital, that 4.1 per cent were women. This is the lowest percentage he found anywhere in the literature, and in his own experience. There is a form of selection implicit in the fact that he is here dealing solely with a group of hospitalized inebriates, which in this country might reasonably be expected to make his percentage lower than the 7.35 shown by the present random sample of the general working class popu-

lation. Etchepare (52) states that in the one insane asylum in Uruguay there were as inmates between 1899 and 1908, 1984 insane women, of whom 52, or 2.62 per cent, were alcoholics (heavy drinkers). He regards this percentage as abnormally low, basing his opinion upon corresponding European data. Minor (53), among 9760 cases of alcoholism treated at the Moscow Dispensary (Ambulatorium), had 1288 women, or 13.5 per cent, between 1903 and 1908. This figure would seem to compare reasonably with our 7.35, considering that probably the Russian population 20 years ago was in general much more intemperate than any American city population in 1918–20. In the 25 years 1886–1910 Petro (54) reports 5029 patients admitted to the insane asylum in Cuneo (Piedmont). Of these 631 (including recidivists) were alcoholics. Thirty-eight, or 6.02 per cent, of these 631 were women. Neglecting the uncertainty about repeaters, this seems to agree quite reasonably with our 7.35. In the present material there were (see Table II) 35 heavy drinkers among the Italians. Of these 3, or 8.57 per cent, were women. But 35 is obviously too small a number to get a reliable percentage from, in such a matter as this. Lombroso (55) quotes from an investigation by Amaldi showing that in a total of 38,764 admissions (22,168 men and 16,596 women) to insane asylums in Italy, in the triennium 1909–1911, 6341 men and 751 women were alcoholics (heavy drinkers). From these figures it appears that of the 7092 heavy drinkers 10.59 per cent were women, and of the 16,596 insane women 4.53 per cent were heavy drinkers. An insane population is not, of course, properly comparable with a sample of a general population. The remarkable thing would seem to be that the percentages in the two cases are as nearly of the same order of magnitude as they are.

The social and biological forces which lie behind these distributions in respect of drinking habits are different in males

and females. In the case of males there can be no question that the pressure of the *mores* (Sumner, 60) in large American cities was preponderantly towards drinking as a habit rather than total abstinence, in the days before national prohibition.[2] This is reflected in the fact that in this material 70 per cent of the males are drinkers, either moderate or heavy. At the same time there has been, in the 60 odd years which in a sense these statistics cover, an unquestionably increasing social pressure against heavy drinking. " Getting drunk " has been more and more frowned upon as a social pastime. In my opinion, and it can only be an opinion, there has not been in Baltimore any particular pressure of the *mores* against moderate drinking *as such,* except from certain of the Protestant branches of religious faith, nor has there been any evident change in this regard during the last 50 years. But the pressure *against* heavy drinking, already spoken of, has in the main, I think, been *towards* abstinence rather than towards moderate drinking. And furthermore there has been the steady pressure of the so-called " temperance " organizations, working chiefly through certain church affiliations, against all use of alcohol as a beverage. In Baltimore the effect of this latter factor does not appear in general to have been great, and in the particular social stratum of the population here dealt with, may probably be regarded as nearly negligible in the case of males.

If the above considerations are in any measure true it would seem probable that the following relations are also true. The group of male abstainers is probably composed broadly of

[2] Whether this has changed appreciably since 1919 is a difficult question, upon which I know of no data adequate to give an answer. My guess is that it is, statistically speaking, still considered by young men more " manly," and certainly smarter, to drink than not to. In any case the point need not trouble us here, because the data in this book relative to alcoholic habits pertain almost exclusively to conditions prior to 1919.

three classes of persons, as follows: (a) Those who do not drink because of fear of the consequences; (b) those who do not drink because they choose to conform to the pressure of opinion, chiefly from family and church (Protestant), against heavy drinking; and (c) those who simply have never had any desire to use alcohol as a beverage. Class (a) may again be divided into two categories. Of these the first group includes those male persons who, knowing or believing that they have a poor constitution, refrain from drinking because they fear that alcohol will injure their health. They regard themselves as weaklings, in greater or less degree, and fear enables them to control their not too strong appetites (note in this connection that Reed's series of nervous and mental cases cited above shows 26.8 per cent of males to have been abstainers). The second category under (a) includes those male persons who fear that they will not be able to control their consumption of alcohol if they start drinking at all. They have, in short, a horror, frequently based upon a sad family experience, of becoming chronic inebriates, and therefore abstain entirely from alcohol. Classes (b) and (c), in the case of males, probably include fewer individuals than class (a), but there is really little existing basis for forming any critical judgment as to the relative frequency of these several classes.

Moderate drinking males may, I think, fairly be regarded as a normal, average group of men, whose health has always been such as to give them no particular concern and who have no misgivings about their power of controlling their appetites. The outstanding feature about them would seem to be their normal, average quality. They are not afraid of drink, or of themselves, on the one hand, nor have they any abnormal or pathological craving for pleasures of the senses, on the other hand. They are, in short, as we experience them in daily life, well-balanced people both mentally and physically. The bio-

logical normality of moderate drinking has been discussed by Kauffmann (56) and by Mercier (57).

Heavy drinkers are preponderantly probably somewhat pathological persons, in the opinion of most students of inebriety. They have a craving, regular or periodic, for an excessive amount of alcohol, which is only with great difficulty ever controllable, and finally comes to be wholly beyond the individual's power to curb.

Becker (61) believes that the tendency to become a drunkard depends upon an innate constitutional abnormality or deficiency.

Astley-Cooper (62) gives the following excellent discussion of the matter (pp. 14–16):

" The following are the principal psychophysical defects and peculiarities found to be present in greater or less degree in all alcoholic inebriates, and on them their inebriety would seem to depend.

" 1. An incapacity to bear physical or mental pain or distress, or an abnormal degree of mental or physical hyperaesthesia, or both. The consequence of such defect or peculiarity is an abnormal need of immediate, adequate, and complete relief of such mental or physical pain or distress at any and all costs.

" 2. Defective moral sense.

" 3. Defective sense of responsibility.

" 4. Intolerance or tolerance of alcohol, below or above normal respectively. The intolerance is seen in periodic and the tolerance in chronic inebriety.

" 5. Defective realization and appreciation of his abnormalities on the part of the inebriate, even when sober, which are obvious to those about him, such defect being observable in a great many persons of unstable mental equilibrium, who are not insane, as well as in insane persons.

" 6. Defective inhibition, making resistance to any real or fancied need, desire or impulse, abnormally difficult. Such inhibition shows itself in relation to most of the affairs of everyday life, and not by any means only in regard to alcohol custom or usage, though such custom long continued increases the defect.

" 7. A generally defective mental equilibrium, showing itself principally perhaps in want of concentration and attention, abnormal emotionalism, unreasonable likes and dislikes, unreasonable and undue self-appreciation or self-depreciation, extremes of optimism and pessimism, cynicism and *tedium vitae*.

" These defects and peculiarities are present in greater or less degree in all pathological inebriates, though not always all present in the same individual, and always in varying proportions.

" Perhaps the defect which varies most is the defect in the moral sense. It is nearly always present in some considerable degree, but may vary very greatly, and is one of the defects which is always made worse by continued alcoholic abuse, and which benefits very much under total abstinence and treatment generally.

" We have now to consider what is the origin of these defects and peculiarities, as such origin will also be the predisposing causes of inebriety.

" There can be no doubt that these defects and peculiarities may be inherent or acquired, and it seems doubtful — very doubtful indeed — whether pathological inebriety can exist unless such defects and peculiarities inherent or acquired are present, and it is equally doubtful whether such peculiarities and defects with the exception, perhaps, of tolerance of alcohol, can be acquired by careless and occasional drinking alone."

Mott (63) in 1911 said: " I am satisfied that the majority of our insane inebriates have become alcoholic because of con-

genital defects or tendency to insanity and not insane as a result of alcoholism, and that the drunkenness which preceded alcoholic insanity was merely the herald, the only obvious sign of mental disorder."

Rogers (58) says of inebriates that "all possess an underlying nervous and mental instability — a something that fails them in times of stress, worry, and storm." Williams (59) also emphasizes the underlying biological abnormality of the drunkard.

Many other authorities might be cited to the same purport.

In the case of females the preponderant social pressure has been against any use of alcohol, I think, in the period covered by these statistics. In total only 32.9 per cent of the women in the present material have been drinkers, whether moderate or heavy. This is as unquestionably a reflection of the *mores* as is the reverse relationship among the males.

Among the abstaining females the same three classes may be recognized as in the males. But the relative magnitude of classes (b) and (c), as compared with (a), is probably larger among females than among males, in the case of the long established German and British elements of the Baltimore population.

In the more recently immigrant "All other" group, composed of Slavonic, Jewish, and Italian stocks, the case is different. Among these people there is much less social pressure towards female abstinence. They are in general less Americanized than the other racial groups, and still retain a large measure of European *mores*. It is not surprising to find among them a slightly larger proportion of moderate drinking than of abstaining females.

The question of the economic status and homogeneity of the material will now be considered. It is possible to classify on this basis 3912 of the 5248 individuals in the total experience.

For the others the information on economic condition is either lacking entirely (mãinly as a result of refusal to furnish it to the field workers) or is stated in general non-quantitative terms, not capable of being reduced to the exact statistical basis necessary here. The sample which is available, however, is large and there is no reason to suppose that it is not random in respect of the item under discussion, namely weekly income in dollars.

Table VIII gives the distribution of the individuals in this sample in respect of the income per week (in dollars) *of the family* to which they belonged, at the time the records were ob-

TABLE VIII

Economic Status of Families

Family income per week (dollars)	Abstainer		Moderate		Heavy	
	Absolute	Per cent	Absolute	Per cent	Absolute	Per cent
0–9	40	2	22	2	13	2
10–19	202	11	215	15	131	19
20–29	597	33	589	41	244	35
30–39	561	31	311	22	189	27
40–49	247	14	139	10	67	10
50–59	126	7	63	4	23	3
60–69	2	0.1	36	3	4	0.6
70–79	24	1	42	3	12	2
80–89	...	...	5	0.4	8	1
Totals.....	1799	99.1	1422	100.4	691	99.6

tained. This means some time between the latter part of 1919 and the first few months of 1922. This was a period of relatively high money wages, following the gross inflation of the war time. In consequence the whole scale of incomes shown in Table VIII is undoubtedly considerably higher absolutely than these same families, or others of the same general rank in the

social scale, would have shown in pre-war times. This fact, however, in no way invalidates the figures for comparison *inter se*. The families represented by the frequencies in the first row of Table VIII (income 0–9.99 dollars) were for the most part receiving charitable aid.

The percentage distributions of Table VIII are shown graphically in Fig. 4.

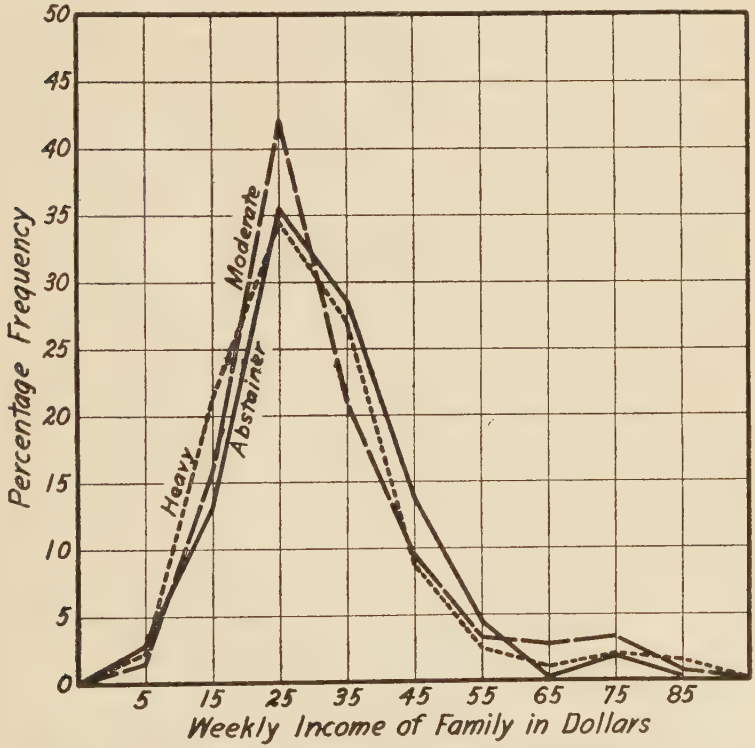

FIG. 4. Frequency polygons showing variation in family income per week. Solid line — abstainers; dash line — moderates; dotted line — heavies.

It is evident from the table and the diagram that there is no great difference in economic status between the persons in

the different drinking habit groups. This is the conclusion which was reached earlier (3) from a similar examination of a smaller sample of the material. The distributions of Table VIII here are like those of Table XI of the study cited.[3]

A better understanding of the degree of differentiation which exists between the three groups in respect of economic status, as indicated by weekly family income, can be got from Table IX, which presents the usual biometric constants derived from the distributions of Table VIII.[4]

TABLE IX

Biometric Constants for Family Income per Week (in dollars)

Constant	Abstainer	Moderate	Heavy
Mean income.....	$32.11 ± .20	$31.35 ± .26	$30.20 ± .35
Standard deviation.....	12.40 ± .14	14.77 ± .19	13.78 ± .25
Coefficient of variation..	38.6 ± .5%	47.1 ± .7%	45.6 ± 1.0%

The average income of above $30 per week may at first thought seem unduly high for a working-class population. By reflection, however, any such illusion will be promptly dispelled. These figures relate to urban families during the war, and immediately post-war, period. Money wages were high.

Comparing the three groups, the statistical results are, in general, of the character which probably would have been expected. The abstainer group shows the highest mean family

[3] The meticulous reader will note that in Table VIII the 4 individuals set down in Table XI of the former study as having a weekly income above $90 have disappeared. This is because a mistake was made, in the former study, about the family to which these individuals belonged. The $90 was a monthly and not a weekly income. They have been properly allocated here.

[4] The reader unacquainted with biometric methods and terminology should consult some elementary text-book of modern statistical methods, such as Yule (65) or Pearl (66), for a discussion of the meaning of these constants.

income per week, and the lowest variability in income, of any group. The moderate group has a mean weekly income 76 cents less than that of the abstainer group, a difference not statistically significant. The moderates, however, are significantly more variable in weekly income, than the abstainers. The heavy group has a mean family income per week which is $1.15 less than that of the moderate group, and $1.91 less than that of the abstainers. The first of these differences is probably not significant statistically, and the second probably is. There is no significant difference in variability in respect of weekly income between the moderate and heavy groups.

It cannot be supposed, with any show of reason whatever, that these small differences in family income per week can possibly explain the differences in duration of life which will presently be shown to exist between the three groups. What Table IX shows is the essential likeness of these drinking groups. The difference of just under $2 per week between the heavy and abstainer groups in average incomes can have no real significance in explaining the higher mortality of the former, for the simple reason that an average family income of $30.20 per week is a long way removed from poverty.

In summary, what I have tried to show in this chapter is that we are dealing in this study with a reasonably homogeneous mass of material, fairly representative of the white working-class population of a large American city. This material has been carefully and honestly collected. It is not so extensive as one would like for a complete statistical discussion of some points. Doubtless critics will point out other deficiencies. Those that I know of I shall discuss in the appropriate places in later chapters. But perhaps I realize more clearly than most of my critics can, just how difficult it is to get trustworthy data about the individual life histories of human beings, on a

statistical mass scale. Within its limitations I know that the present material has a degree of accuracy unusual in statistical data of similar character for man. Perhaps its shortcomings in other directions will in some measure be excused on this account.

CHAPTER III

THE CLASSIFICATION OF THE MATERIAL

Obviously the first necessary step in the statistical treatment of the material is to classify the individuals in respect of their drinking habits. This is, on the face of it, a difficult thing to do. Not only is a consumption of alcohol which is regarded by one man as moderate looked upon by another as heavy drinking, but in real fact it may be. There is great individual variation in susceptibility to ethyl alcohol among human beings. Cadbury (68) describes a patient who regularly showed symptoms of intoxication on taking 2.85 c.c. of alcohol in 15 c.c. of water. On the other hand consider such a person as the famous Dr. George Fordyce, a distinguished eighteenth century physician, public lecturer and scientist, of high repute in all these fields. He was a Fellow of the Royal Society and of the Royal College of Physicians, and a member of the famous Literary Club to which Dr. Johnson belonged. For above twenty years he maintained unswervingly the following *daily* regimen (I quote from Timbs (69) pp. 288–289 — the facts are well known and recorded in a number of other places):

"At four o'clock, his accustomed dinner hour, he entered Dolly's chop-house, and took his seat at a table always reserved for him, on which were instantly placed a silver tankard full of strong ale, a bottle of port-wine, and a measure containing a quarter of a pint of brandy. The moment the waiter announced him, the cook put a pound-and-a-half of rump-steak on the gridiron; and on the table some delicate trifle, as a *bonne bouche,* to serve until the steak was ready. This deli-

cacy was sometimes half a boiled chicken, sometimes a plate of fish; when he had eaten this, he took a glass of his brandy, and then proceeded to devour his steak. We say devour, because he always ate rapidly as if eating for a wager. When he had finished his meat, he took the remainder of his brandy, having, during his dinner, drunk the tankard of ale, and afterwards the bottle of port.

"The Doctor then adjourned to the Chapter Coffee-house, in Paternoster Row, and stayed while he sipped a glass of brandy and water. It was then his habit to take another at the London Coffee-house, and a third at the Oxford, after which he returned to his house in Essex Street, to give his lecture on chemistry. He made no other meal till his return next day, at four o'clock at Dolly's."

There, indeed, was a man!

Kabrel (70) discusses thoughtfully, but without definite supporting evidence, the variation among individuals in susceptibility and tolerance to alcohol, from the point of view of dangerous habit formation, and concludes that because of this variability it is impossible to regard any particular quantity as a "safe" dose of alcohol. Becker (71), however, reaches a somewhat different conclusion as the result of an investigation of considerable originality and interest which he carried out. He collected, mainly through physicians, detailed biographies of 50 people — 28 men and 22 women from all walks of life — all of whom were in their ninetieth year or older. From the data as to the habits of these people Becker calculated the average daily dose of absolute alcohol consumed by each throughout life. He then argues that even the greatest of these averages must be regarded as a non-toxic dose, indeed well within the margin of safety, because surely there can be no talk of an artificial cutting off of life by bad habits in the case of persons who live to be 90 or more. The calculations

were based upon the amounts consumed during the major portion of the subjects' lives, and not upon their habits in extreme old age. Becker points out that it is reasonable to suppose that whatever damage to the system was going to be done by alcohol, would have been fairly well accomplished by the time age 70 was reached. In 41 cases the information was sufficiently accurate and detailed to be usable. Of the 41 persons 3 men and 4 women were total abstainers, and hence brought down the average for the whole group. This average, based on the whole 41, was 14.86 grams *per diem* of pure ethyl alcohol. Becker concludes that so far as health and longevity are concerned it is not in any way radical to suppose that at least 15 grams of alcohol (properly diluted and flavored, of course, as normally in beverage alcohol) may be regarded as a safe daily dose.

In attempting to classify persons relative to their drinking habits, for the purpose of investigating the effect of such habits upon health and longevity, there must first of all be some rigorously defined concept as to differences in degree of consumption. It will serve no particular useful purpose to contrast all drinkers with total abstainers. It is precisely this error which makes those insurance data on the problem, which would otherwise be worthy of credence, largely nugatory. It implies that the question to be answered is: Does drinking shorten life? This is not the problem at all. This question is on the face of it essentially a silly one, for the plainest of facts of experience is that " drinking " is not a unique category. There are many different kinds and degrees of drinking. What a sensible person wants to know is whether *all* the different kinds and degrees of drinking are prejudicial to health and longevity, and if not, which ones are and which ones are not, and to what extent for each of the different categories. Therefore in setting the stage for a real scientific analysis of the problem the

first requisite is to define as accurately as possible the different categories of drinking habits which in fact exist.

As was promptly pointed out by my English critics (29) I fell in some part into the error discussed above, in my first treatment of the problem (49). I there grouped the individuals into three categories in respect of drinking habits, as follows:

1. Total abstainers.
2. Moderate and occasional drinkers.
3. Heavy or steady drinkers.

These classes were then discussed as follows (pp. 243–244):

" The class ' Total abstainers ' includes only such persons as were known never to have used alcoholic beverages at all, even in the smallest amounts. If any person occasionally took a glass of beer, or wine, he went into the class of ' Moderate and occasional drinkers.'

" The second class, ' Moderate and occasional drinkers,' includes all those persons whose drinking habits, in the opinion of family and friends, were very moderate in respect of amount consumed and not entirely regular or steady in respect of frequency. Here were included persons who ' took an occasional glass of beer,' the labourer who at irregular and not too frequent intervals took a drink of whiskey, or other spirits. The person who made it a regular habit to take wine or beer with meals, even though the amount so taken was never excessive, was placed in the next higher class, the ' Heavy or steady drinkers.' This was done on the ground that a *steady* daily drinker, even in moderate amounts at any one time, consumes in the course of a lifetime a good deal of alcohol. Furthermore, there are in his life-history no periods measured in units of time greater than hours, in which his system is not metabolising some alcohol. I realize fully that in placing the moderate and temperate but *steady* daily drinker in the ' Heavy ' category that I am going contrary to common opinion and the

common usage of descriptive language. But I believe that the classification here adopted is more nearly scientifically warranted in an objective study of the problem than is that based upon common opinion.

" The third class, ' Heavy or steady drinkers,' includes all of those whose habits are described by relatives and friends as coming under either of these categories. It includes the drunkards, on the one hand, and the steady daily drinkers who never or rarely at any one time drink to such excess as to become intoxicated."

In the second edition of the English Committee's book *Alcohol* (29) the following remarks are made about this classification (pp. 141–142):

" Naturally an investigator is entitled to group his data into whatever classes he thinks appropriate, provided the definitions of the grouping be unambiguous. We can therefore merely express our regret that Prof. Pearl has adopted a method of classification which deprives us of nearly all the benefit which we had hoped to derive from his admirable material.

" We should not indeed have supposed that the most convinced advocate of total abstinence would have maintained that the occasional use of alcoholic beverages in a manner qualifying for admission to Prof. Pearl's second class would be likely to shorten life to an extent capable of being statistically demonstrated. Indeed the answer to such a question seems to us of hardly any practical importance. The really serious question, the one which, as we have pointed out, Assurance data do not enable us to answer in any convincing manner, is whether moderate drinking, using the word moderate in its colloquial sense, is prejudicial to longevity. Are a daily pint or two of beer, or a daily bottle of claret, or a few glasses of whiskey and soda *per diem* harmful? Prof. Pearl's analysis, if we have correctly interpreted his method of classification, throws

no light at all upon this fundamentally important practical point. His third class contains in unstated proportions those who in general usage would be termed 'moderate' and those who would be termed 'heavy drinkers.'"

I admit fully the force of this criticism, and because I do, have in the present volume adopted a different classification, presently to be set forth. At the same time I am not prepared to admit that the logic behind the first classification was not sound from a purely biological point of view. The real difficulty with it arises from the fact that all men are not guinea-pigs, and cannot be treated as if they were in a matter of this kind. In short, in a matter in which the variables concerned are longevity and alcohol, the problem is complicated by the important rôle played in the case of man by factors not directly and purely biological. For this reason it is my own opinion (in which I do not ask anyone to concur) that carefully conducted experiments with animals will in the long run produce more reliable and trustworthy evidence as to the effect of alcohol, as such, upon duration of life as such, including human life, than will any human data.

The classification upon which the material will be treated in the present volume was drawn up expressly to meet the strictures of the English critics. I am assured that it does so. The present classification includes eight groups, as follows:

1. Abstainers.
2. Moderate drinkers in respect of the amount of alcohol consumed, without specification as to the regularity or frequency of their drinking.
3. Moderate in respect of the amount consumed, occasional as to frequency.
4. Moderate in amount, steady as to frequency.
5. Heavy in respect of amount of alcohol consumed, without specification as to the regularity or frequency of drinking.

6. Heavy in amount, occasional as to frequency, and abstainers in the intervals between heavy drinking bouts.

7. Heavy in amount, occasional as to frequency, and moderate drinkers in the intervals between heavy drinking.

8. Heavy in amount, and steady as to frequency.

As to these categories the following may be said. An abstainer is a total abstainer who has never used alcohol in any form. A moderate drinker is one who uses alcohol in any form (beer, wine, or spirits), but in small amount at any one time and never enough to become intoxicated. The persons put in the moderate "steady" category are those whose habits are precisely of the sort described in the British Committee's report (*loc. cit.*, p. 141): "A daily pint or two of beer, or a daily bottle of claret, or a few glasses of whiskey and soda." The moderate "occasional" class includes those who drink to this amount, but not as frequently or regularly as every day. The moderate "unspecified" class includes those of whom it was definitely known that the amount consumed fell in the moderate category, but regarding whom the record was incomplete or indefinite as to frequency. The heavy class includes those persons who are positively known to have been in the habit of getting drunk; either as an occasional spree, with abstinence in the intervening periods (heavy, occasional, otherwise abstainer); or with moderate drinking as above defined in the intervening periods (heavy occasional, otherwise moderate); or regularly and frequently (heavy, steady); or without precise record of the frequency of intoxication (heavy, unspecified).

I believe that this classification fairly meets the criticism of the British report. Surely it is in accord with common usage to call a person who gets drunk a heavy drinker. Also it is common usage to call a person who drinks a little but never gets drunk a moderate drinker. Obviously categories 2 and

5 are products of necessity solely. They include those individuals for whom the information extends only to amount and not to frequency.

It seems desirable to quote here a few of the original records as to drinking habits in order that the nature of them may be seen, and that the application of the classification may be understood. The following records are taken at random where the finger fell after turning over casually bundles of pages of the record book.

Case 33, No. 8. Male. Dead at 25 years of tuberculosis. "Drank a great deal. Often intoxicated." Classified as *Heavy, steady.*

Case 55, No. 11. Male aged 59. "Drank whiskey and beer. Would get drunk 4 or 5 times during the year; other than that would drink beer with his meals and whiskey occasionally." Classified as *Heavy, occasional, otherwise moderate.*

Case 71, No. 43. Male aged 38. "Beer with meals, sometimes a little wine." Classified as *Moderate, steady.*

Case 77, No. 20. Male aged 52. "Never drank steadily. Goes on sprees about once or twice a year." Classified as *Heavy, occasional, otherwise abstainer.*

Case 82, No. 14. Female aged 35. "Drinks beer at times, mostly at holidays. Whiskey very little." Classified as *Moderate, occasional.*

Case 147, No. 11. Male. Dead at age 84. "Drank beer with his meals, one or two large glasses. One or two glasses before retiring." Classified as *Moderate, steady.*

Case 150, No. 23. Male aged 38. "Likes to drink and drinks 'right often.' Says he drinks a lot too. Drinks now as much as he did before prohibition — says he can get it whenever he wants it." Classified as *Heavy, steady.*

Case 169, No. 10. Male. Dead at age 44. "Began to drink when 25 years old. Would sign a pledge for 3 or 5

years and when time would expire would go on sprees which would last a week or 10 days." Classified as *Heavy, occasional, otherwise abstainer.*

Case 187, No. 6. Male. Dead at age 36. "Drank beer, wine and whiskey, or anything he could get; got drunk regularly every Saturday and Sunday." Classified as *Heavy, steady.*

Case 209, No. 474. Male. Dead at 59. "Began drinking when 25 years old. Would go on sprees once a week or every two or three weeks, but not so as to have to stop his work. Has gone as long as 7, 8, or 9 days without drinking at all. Did not use alcohol at all last 3 or 4 years of life." Classified as *Heavy, occasional, otherwise moderate.*

It is obvious that essentially the basis of the present classification is the same as that which we use regarding drinking in everyday life. It is that of the immediate *effect* of the drinking on the drinker. If a person drinks enough to get drunk it is *prima facie* evidence that he is a heavy drinker. In addition to its conformity to the actualities of life this basis has the further advantage that the information obtained in the way this was will be generally reliable. It only involves the conceptual definition of intoxication. And in the class of people with whom we are here dealing there is no doubt or reservation as to the meaning of "drunk." They perfectly well know when a person is drunk. They would have scant courtesy for such a definition as that of Kelly (72), who argues that any person that takes any quantity of alcohol of any dilution is *ipso facto* intoxicated, because alcohol is a toxic substance. Such pedantry would only be a hindrance in any attempt like the present one to determine scientifically the influence of alcohol upon duration of life. The concept of drunkenness upon which the present classification is based is much closer to that stated by Charpentier (73) to have been

the one long employed by the London police, namely "A man is drunk (in a police sense) if he is so under the influence of alcohol as to be a nuisance or a danger to himself or others."

The number of persons in each of the drinking categories is shown in Table X.

TABLE X

FREQUENCY OF PERSONS IN THE SEVERAL CATEGORIES OF DRINKING HABITS

Drinking Habits	Males			Females		
	Absolute	Per cent as to habits	Per cent as to sex	Absolute	Per cent as to habits	Per cent as to sex
Abstainers	926	100	39	1452	100	61
Moderate in amount:						
Unspecified as to frequency	325	25	86	55	8	14
Occasional	439	33	54	367	57	46
Steady	549	42	71	223	35	29
All moderate	1313	100	67	645	100	33
Heavy in amount:						
Unspecified as to frequency	409	48	92	37	55	8
Occasional, otherwise abstainer	30	4	94	2	3	6
Occasional, otherwise moderate	105	12	96	4	6	4
Steady	301	36	93	24	36	7
All heavy	845	100	93	67	100	7
Total	3084	...	59	2164	...	41

Approximately three-fifths of the total abstainers are women and two-fifths are men. Two-thirds of the moderate drinkers are men and one-third women. But among the moderate occasionals men and women occur in nearly equal numbers. In the moderate unspecified class 86 per cent are men. This merely indicates the greater frequency of comprehensive information about drinking habits among the females. In all the heavy drinking classes the sex distribution is about the

same. Roughly 93 per cent of heavy drinkers in this experience are men.

Turning to the other columns of percentages it is seen that among the moderate drinkers one-third of the men and over one-half of the women are occasional as to frequency. Nearly the same percentage of women as of men are moderate steadies. Among the heavies the distributions of men and women are surprisingly similar, so far as concerns the two occasional and the steady groups. Exactly the same percentage, namely 36, of heavy drinking men and heavy drinking women are steady as to frequency.

We may turn next to the consideration of an important and interesting phase of the subject. What of the persons who change their drinking habits during life, in a definite, permanent, and often radical way? Such persons constitute a special methodological problem in the study of the effect of alcohol upon life duration. So far as I am aware no attempt has been made in previous work on the subject to deal with these persons separately. The present records make possible a definite segregation of them.

Table XI presents the pertinent data about all persons in the present record who definitely and permanently changed their drinking habits in the course of their lives, up to the date of the record, or to death.

The person who changes his drinking habits in a vacillating manner really does not change them at all. The toper who "swears off" for a few days, or weeks, or months, or years, and then resumes his heavy drinking, and repeats this performance again and again till he dies, simply has vacillation as an integral part of his habits relative to drink, and is to be classified accordingly. We are concerned in Table XI with quite another category of persons, namely those who change their drinking habits, and stay changed.

TABLE XI

DATA ON CHANGES IN DRINKING HABITS IN THE PRESENT MATERIAL

Original category	Age at change	Subsequent category	Years under second category	Frequency, sex and status
Moderate, unspecified	40	Abstainer	4	1 ♂ D
Moderate, occasional	60	"	10	1 ♀ L
" "	60	"	15	1 ♂ D
Moderate, steady	25	"	20	1 ♂ L
" "	25	"	3	1 ♂ L
" "	25	"	20	1 ♂ L
" "	25	"	14	1 ♂ D
" "	30	"	9	1 ♂ L
" "	30	"	32	1 ♂ L
" "	50	"	8	1 ♂ L
" "	50	"	15	1 ♂ L
Total moderates shifting to abstainers	38.2	D = 41.7 + = 52.7 D = 11.0 L = 36.9 + = 51.5 L = 14.6	13.6	10 ♂ 1 ♀
Heavy, unspecified	?	Abstainer	?	1 ♂ L
Heavy, steady	25	"	8	1 ♂ L
" "	30	"	16	1 ♂ L
" "	30	"	1½	1 ♂ D
" "	35	"	5	1 ♂ L
" "	35	"	24	1 ♂ L
" "	35	"	30	1 ♂ L
" "	37	"	34	1 ♂ L
" "	40	"	20	1 ♂ L

TABLE XI (continued)

Original category	Age at change	Subsequent category	Years under second category	Frequency, sex and status
Heavy, steady	40	Abstainer	1	1 ♂ D
" "	40	"	5	1 ♂ D
" "	40	"	46	1 ♂ D
" "	45	"	7	1 ♂ D
" "	45	"	4	1 ♂ L
" "	50	"	9	1 ♂ L
" "	50	"	13	1 ♂ L
" "	50	"	17	1 ♂ L
" "	50	"	34	1 ♂ L
" "	50	"	20	1 ♂ D
" "	50	"	34	1 ♂ D
" "	55	"	4	1 ♂ D
" "	67	"	4	1 ♂ L
Total heavies shifting to abstainers	42.8	D = 43.75 + = 58.55 D = 14.8 L = 42.2 + = 59.0 L = 16.8	16.0	22 ♂
Moderate, steady	41	Moderate, occasional	6	1 ♂ D
Heavy, steady	27	Moderate, unspecified	20	1 ♂ L
" "	30	" "	8	1 ♂ L
" "	30	" "	12	1 ♂ L
" "	30	" "	26	1 ♂ L
" "	30	" "	30	1 ♂ L
" "	30	" "	38	1 ♂ L
" "	30	" "	17	1 ♂ D

TABLE XI (continued)

Original category	Age at change	Subsequent category	Years under second category	Frequency, sex and status
Heavy, steady	30	Moderate, unspecified	19	1 ♂ D
" "	30	" "	43	1 ♂ D
" "	30	" "	40	1 ♂ D
" "	30	" "	54	1 ♂ D
" "	30	" "	55	1 ♂ D
" "	45	" "	6	1 ♂ D
" "	60	" "	15	1 ♂ L
" "	60	" "	15	1 ♂ L
" "	60	" "	9	1 ♂ D
" "	60	" "	15	1 ♂ D
" "	63	" "	2	1 ♂ D
" "	50	Moderate, occasional	22	1 ♂ D
" "	55	" "	20	1 ♂ D
" "	68	" "	10	1 ♂ L
Total heavies shifting to moderate	41.8	D = 37.75 + = 62.95 D = 25.2 L = 40.6 + = 59.5 L = 18.9	22.7	21 ♂
Total shifting from more to less drinking	41.4	D = 42.9 + = 62.0 D = 19.1 L = 40.3 + = 57.6 L = 17.3	18.1	54 ♂ 1 ♀
Abstainer	60	Moderate, steady	7	1 ♂ D
Abstainer	30	Heavy, steady	26	1 ♂ L
"	30	" "	5	1 ♂ D
"	40	" "	10	1 ♂ L
"	40	" "	21	1 ♀ D
"	50	" "	23	1 ♂ D
"	50	" "	10	1 ♂ D
Total abstainers shifting to heavy steady	40.0	D = 42.5 + = 57.25 D = 14.75 L = 35.0 + = 53.0 L = 18.0	15.8	5 ♂ 1 ♀

TABLE XI (concluded)

Original category	Age at change	Subsequent category	Years under second category	Frequency, sex and status
Moderate, unspecified	20	Heavy, steady	10	1 ♂ L
" "	20	" "	17	1 ♂ D
" "	23	" "	15	1 ♂ D
" "	25	" "	2	1 ♂ L
" "	25	" "	10	1 ♂ D
" "	25	" "	23	1 ♂ D
" "	26	" "	4	1 ♂ D
" "	28	" "	20	1 ♂ L
" "	28	" "	2	1 ♂ D
" "	40	" "	10	1 ♂ L
" "	40	" "	2	1 ♂ D
" "	40	" "	7	1 ♂ D
" "	40	" "	10	1 ♂ D
" "	40	" "	14	1 ♂ D
" "	60	" "	28	1 ♂ D
" "	63	" "	15	1 ♀ D
Moderate, occasional	72	" "	1	1 ♂ D
Moderate, steady	32	" "	2	1 ♂ D
" "	35	" "	3	1 ♂ D
Total moderates shifting to heavy	35.9	D = 37.9 + = 48.1 D = 10.2 L = 28.25 + = 38.75 L = 10.5	10.3	18 ♂ 1 ♀
Total shifting from less to more drinking	37.8	D = 39.95 + = 50.9 D = 10.95 L = 30.5 + = 43.5 L = 13.0	11.4	24 ♂ 2 ♀

The first point to be discussed from this table is the relative frequency of persons who definitely changed their drinking habits in this experience. Out of 1313 moderate drinking males (in Table X) only 10, or 0.8 per cent, became total abstainers. On the other hand 22 heavy drinking males out of a total of 845 such persons in the experience became abstainers, a percentage of 2.6, or relatively more than three times as many as made the change from moderate drinking to total abstention. This difference in the two cases is what would be expected, I think. There is an enormously greater incentive to stop heavy drinking than there is to stop moderate drinking. One female, out of a total of 645 moderate drinkers of that sex, became an abstainer, 0.2 per cent. One moderate steady male reduced to moderate occasional out of a total of 549, again approximately 0.2 per cent. Among 301 heavy steady male drinkers 21, or 7.0 per cent, changed to moderate drinking. Taking the whole male drinking group together, regardless of the degree of their drinking, there are in this experience 2158 persons. Of these 54, or 2.5 per cent, changed in the course of their lives up to the time of the record, or of death, from more to less drinking. This proportion will doubtless seem small. It almost certainly is, I think, an understatement of the case for the general population, and perhaps is even for the working-class population from which the present material is drawn. I do not feel sure, however, that the latter point is true. The workingman is, I think, much less apt to change his drinking habits in the direction of a reduction, in the absence of compulsion, than is the man in the higher social and economic strata of society. Why should he? As many writers on social and psychological matters have pointed out, alcohol is one of the very few reliable sources of the enjoyment which comes with the letting down of nervous tension, which are available to the poor man. That he may,

and too often does, abuse it is beside the point in the present connection. If he has once become impressed with its reliability to produce what he regards as an enjoyable state he does not lightly relinquish such a resource. Incidentally it may be remarked that a striking confirmation of the truth of this contention is afforded by the enormous prevalence of home-brewing, wine-making, and distilling among the working-class population of large American cities at the present time. The wealthier and less irresponsible professional and business man has more foresight than the workingman, statistically speaking, and much more frequently, I think, convinces himself on rational grounds that a shift from more to less drinking is desirable, and acts upon this conviction.

Turning now to the consideration of changes in drinking habits in the opposite direction, that is from less to more drinking, the following relations appear. One male abstainer out of a total of 926 such persons changed to a moderate drinker at the age of 60, and 5 more abstainers, or approximately 0.5 per cent, changed to heavy drinkers at 30 years of age or over. The smallness of these figures is mainly occasioned by the fact of the age limit set in respect of changes from total abstention to drinking. Obviously drinking must have *begun* in all drinkers at some time (age) or other. For reasons which will more fully appear as we go on, I have made it a rule in dealing with this material to regard the beginning of drinking under age 30 as the *starting* of a habit, rather than the *change* of a habit. What the figures above cited show, then, is the number of persons who, having been abstainers up to 30 years of age, thereafter change their habits as to drinking. From general experience of people and of life this proportion would be expected to be small, and so it is. In this group is one female, who, having been an abstainer up to 40, then became a heavy drinker.

Of the 1313 moderate males 18, or 1.4 per cent, changed to heavy steady in their habits. One female also made the same change. Altogether there are 24 males out of a total of 2239, or 1.1 per cent, who changed definitely from less to more drinking as an habitual matter, under the specifications regarding the material set forth above. This result again will seem too small to some. I am confident, however, that it represents substantially the fact, so far as the present material is concerned. Furthermore it seems to be in accord with what is known generally of the conservatism and constancy of habits generally, when they are once firmly established.

We may next consider the age at which the change in drinking habits occurred. Taking all persons together, both living and dead at the time of record, it is seen that the 55 who shifted from more to less drinking make the change at an average age of 41.4 years. The 26 persons making a definite shift in the opposite direction, from less to more drinking, did so at an average age of 37.8 years, or 3.6 years younger. With such small numbers involved this difference probably cannot be regarded as significant. Indeed, with the exception of the last group in Table XI, the moderate changing to heavy drinking, the striking thing is the closeness with which the average ages of the different groups, at the time of making a change in drinking habits, hover about 40 years.

When the dead are separated from the living in this experience the average age at which the change in drinking habits occurred is generally higher in the dead. The only exception is found in the group of heavies shifting to moderate drinking. The general result on this point is that which would be expected. Those persons who were in general older when they made a change in drinking habits would be likely to show a greater mortality in the interval between that time and a later fixed date (the time of the record) than would a group younger

at the start, for the simple reason that all specific death rates increase with age after childhood is passed.

It is of great interest to examine the figures for the average duration of survival after the change in drinking habits. Here the data from those dead at the time of record will obviously be first considered. Confining attention to these it is seen, first, that the dead heavies who became abstainers at an average age of 43.75 years survived, on the average, 14.8 years thereafter, while the dead moderates who became abstainers at 41.7 years survived only 11 years thereafter. The numbers involved are too meager to warrant any definitive conclusions about the meaning of this difference. Indeed it may be entirely accidental. From Glover's (74) life table for white males in cities of the original registration states in 1910 it appears that the average after lifetime ($\mathring{e}_x$) at age 42 is 23.94 years, and at age 44 is 22.57 years. Obviously 11.0 and 14.8 are much smaller than these figures. One must not, however, rashly conclude that this difference marks an impairment of life expectation by the drinking prior to age 40, for the reason that the group of "dead before time of record" persons are selected because they died. Naturally their mean after-life time after about 40 would be expected to be smaller than that of the general life table. The only proper way in which to test the matter is to use all the experience, including both those who died before the record was taken (D) and those who were living at that time (L), and determine the true death rates (q_x). This I have done, by the standard actuarial methods described further on, for the 33 persons in the two groups under discussion. Naturally the results with such a small number cannot be anything but rough, but they will be less misleading than the average survival values of Table XI. The observed q_x values were unexpectedly smooth with one or two exceptions. In view of the small number of cases I have attempted nothing more than

a graphical smoothing. The results are exhibited in Table XII. In this table are also put the corresponding q_x values from Glover's life table cited above, in order that a comparison may be made.

TABLE XII

Life Table Death Rates per 1000, for a Group of 33 Persons (of whom one was a Female) who Changed from Moderate or Heavy Drinking to Total Abstinence at an Average Age of about 40

Age	1000 q_x		
	Observed	Smoothed	Glover
30	6.5	6.5	7.22
35	6.8	7.0	9.73
40	15.3	8.0	12.10
45	9.3	9.5	15.18
50	10.3	12.0	19.17
55	11.4	15.0	26.93
60	0	22.0	38.51
65	0	30.5	53.66
70	37.0	42.0	74.20
75	62.5	57.5	105.46
80	69.0	77.0	145.88

It is now apparent that so far from this group of individuals having a heavier mortality than normal, as one might infer from the average survival figures of Table XI, their death rates at all ages from 35 on to 80 are distinctly *lower* than the corresponding normal life table rates. Especially is the superiority marked after age 45.

The most interesting comparison in respect of the mortality (or duration of life as one pleases) after definite and permanent changes in drinking habits, will be that between the group of 55 persons who changed from more to less drinking at an average age of 41.4 years, with the 26 persons who changed from less to more drinking at an average age of 37.8 years.

The results of such a comparison, by the same method as has just been discussed, are shown in Table XIII.

TABLE XIII

LIFE TABLE DEATH RATES PER 1000, FOR (*a*) 55 PERSONS WHO CHANGED FROM MORE TO LESS DRINKING, AND (*b*) 26 PERSONS WHO CHANGED FROM LESS TO MORE DRINKING

Age	1000 q_x				Glover
	Changed to less drinking		Changed to more drinking		
	Observed	Smoothed	Observed	Smoothed	
30	3.8	5.0	35.6	10.5	7.22
35	3.9	5.5	43.0	14.5	9.73
40	8.6	7.0	12.3	19.5	12.10
45	19.9	8.0	29.0	27.0	15.18
50	11.5	10.5	43.0	37.5	19.17
55	6.3	14.0	0	50.5	26.93
60	0	20.0	70.2	69.0	38.51
65	19.8	29.0	90.9	100.0	53.66
70	40.5	46.0	153.5	150.0	74.20
75	115.9	65.0	250.0	210.0	105.46
80	81.6	90.0	.....	.....	145.88

The three smoothed columns of Table XIII are shown graphically in Fig. 5.

The results shown in Table XIII and Fig. 5 seem striking, but it must be remembered that the exposure to risk is small, and in consequence the raw q_x values are irregular, and too much weight cannot be given to their indications. Whether we take observed or smoothed values it appears, taking the figures at their face value, that those persons who definitely reduced their drinking (from more to less) in general enjoyed a definitely lower mortality at all ages than did the comparable part of the general population. On the other hand those who at some time

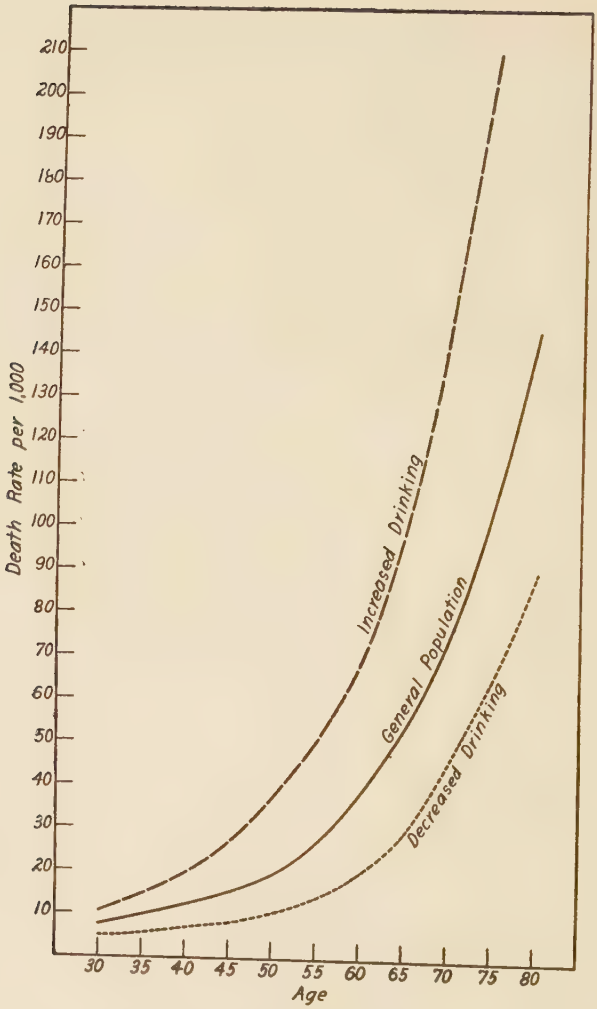

FIG. 5. Life table death rates for (a) the general population (white males in cities of the Original Registration States, 1910) shown as a heavy solid line; (b) persons who definitely increased their drinking during life, shown by a dash line; (c) persons who definitely decreased their drinking during life, shown by a dotted line.

in life definitely increased their drinking in general had a higher mortality than did the general population. The effect of reducing drinking upon expectation of life appears to be just as marked in the passage from heavy to moderate, as it is from heavy to abstention. This tends to increase one's suspicion that the experience is too small to warrant definite conclusions. It is unfortunate that this experience in respect of persons who definitely and permanently changed their drinking habits is not large. But even as it is I think that the indication afforded of the harmful effects upon longevity of heavy drinking, and of the beneficial effects of a reduction of drinking is interesting in a suggestive, if not a final way.

In the earlier work upon the material each of these persons who changed their drinking habits was assigned to a single drinking category, and included in the life tables for those categories. In making these assignments account was taken of all the available information about the person, and an attempt was made to put him in that category which most nearly represented the average of his drinking habits over his lifetime. But plainly it is more exact to leave these persons entirely out of the life tables based on unchanging categories. This has been done in the life tables presented in later chapters of this book for unchanging drinking categories. As a matter of fact no essential or significant difference was made in the result by doing this. The number of changing persons is too small to affect significantly the group results except in certain groups which are themselves too small to warrant life table construction in any case.

In support of this statement Table XIV is presented. This table shows the age distribution and status (living L, or dead D) of the 81 persons under discussion, ranged under the *single* drinking categories to which they were assigned on the basis stated above.

TABLE XIV

AGE DISTRIBUTION AND STATUS OF PERSONS WHO CHANGED THEIR DRINKING HABITS, ACCORDING TO THE SINGLE DRINKING CATEGORIES TO WHICH THEY WERE ASSIGNED

Age group	Moderate unspecified		Moderate occasional		Moderate steady		Heavy unspecified		Heavy occasional, otherwise abstainers		Heavy occasional, otherwise moderate		Heavy steady	
	D	*L*	*D*	*L*	*D*	*L*	*D*	*L*	*D*	*L*	*D*	*L*	*D*	*L*
15–19	..	..	..	..	..	..	..	..	..	..	..	..	..	..
20–24	..	..	..	..	..	..	..	..	..	..	..	..	..	..
25–29	..	..	..	..	..	1	..	1	..	..	..	..	..	..
30–34	..	..	..	..	..	..	..	..	..	1	4	1	1	..
35–39	..	..	..	1	1	..	1	..	..	..	1	1	2	..
40–44	1	..	..	..	..	..	..	..	..	1	1	1	1	..
45–49	..	..	..	2	1	1	1	..	2	2	..	1	2	1
50–54	..	..	..	..	..	..	..	..	1	1	2	..	1	1
55–59	..	..	..	..	..	1	..	1	..	1	1	1	..	..
60–64	..	..	..	1	..	..	1	1	1	..	..	1	..	1
65–69	..	..	1	2	..	..	1	2	..	2	1	..	1	..
70–74	..	..	..	1	..	..	1	..	2	..	2	..	..	1
75–79	..	..	1	..	..	..	..	..	..	..	4	2	..	1
80–84	..	..	..	..	..	..	..	..	1	1	1	..	..	..
85–89	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Totals	1	0	2	7	2	3	6	5	8	9	17	8	8	5

From this table it is seen that of the 325 moderate unspecified males in the experience, only 1 was derived from this group in which the habits were changed during life. Of the 439 male moderate occasionals 8, or 1.8 per cent only, came from the changing group and of the 367 females assigned to the same category only 1 came from this group. Of the 549 moderate steady males only 5, or 0.9 per cent, came from the group under discussion. Of the 409 heavy drinking males unspecified as to frequency 11, or 2.7 per cent, came from the changing groups.

So far these persons who changed their drinking habits obviously form too insignificant a proportion of the final single drinking category group, to which they were assigned, to affect sensibly the actuarial computations based upon those groups. In the next two groups, however, which are the smallest in the whole experience anyway, the case is different. Of the 30 heavy occasional otherwise abstainer males 16, or 53.3 per cent, were persons who changed their habits during life, and of the 2 women in this category 1 belonged to the changing group. As a matter of fact, all that this means is that this single category (and the one following it in Table X) was set up primarily to take care of just the kind of history presented by certain of the persons who changed their habits during life. Of the 105 heavy occasional otherwise moderate males 24, or 22.9 per cent, were from the changing group, as was also 1 of the 4 females in the same category. Since, however, the total numbers in both of these heavy occasional categories are too few to warrant separate life tables, no difficulty is created by putting in these categories the changing drinkers.

Finally it appears that of 301 heavy steady males 13, or 4.3 per cent, came from the changing group, again a small proportion.

It will further appear from data presented in a later chapter

that the age distributions of these changing drinkers, as given in Table XIV, are not essentially different from those of the remainder of the persons in the single drinking categories to which they were originally assigned.

But in the interests of meticulous accuracy, and to forestall at least one of the innumerable criticisms certain to be directed against any investigation of the effects of alcohol upon the organism, all of these 81 persons who changed their drinking habits have been omitted from the experience in the calculation of the life tables according to drinking categories.

CHAPTER IV

ACTUARIAL METHODS AND CHARACTERISTICS

Methods

In the preceding chapters the nature of the material on which this study is based, its mode of collection, and its classification have been described in detail. It remains, as the final step in the discussion preliminary to the exposition of the results, to describe the actuarial methods used in the treatment of the material.

As there has been alleged to have been some misunderstanding of what I thought was a perfectly clear statement on the point in earlier papers (75 and 76) on this material, I wish to say categorically that in the construction of all the life tables in this book, without any exception or reservation whatsoever, *the exposure to risk has been calculated on the basis of all the persons in the experience belonging to the category under discussion, including both those living at the time of the record and those dead at that time.* Table XV shows the age distribution of the persons comprising the total experience, on whose lives the actuarial determinations to be exhibited later were made, and shows further whether they were alive or dead at the time when the record was made. The ages stated are, in the former case, those at the time of record, and in the latter case at death.

The total number of persons shown in this table is 5246. Two persons were unrecorded as to status (whether living or dead). These bring the total to 5248, the same as that of Table II *supra.*

TABLE XV

SHOWING THE NUMBER OF PERSONS IN THE EXPERIENCE, BY AGE, SEX, AND STATUS (WHETHER LIVING OR DEAD AT TIME OF RECORD)

Age in years	Males		Females	
	Dead	Living	Dead	Living
15–19	29	180	21	132
20–24	44	179	41	148
25–29	85	150	47	132
30–34	114	153	49	121
35–39	120	141	58	143
40–44	132	136	51	115
45–49	110	139	45	108
50–54	147	106	75	96
55–59	113	82	60	58
60–64	107	89	67	63
65–69	131	65	88	70
70–74	141	49	75	53
75–79	108	30	81	33
80–84	75	18	41	18
85–89	40	7	34	7
90–94	27	2	14	8
95–99	11	3	5	2
100–104	2	1	..	..
Age unknown	12	4	3	2
Totals	1548	1534	855	1309

The data of Table XV concerning the living persons in the present experience are put in percentage form in Table XVI, and for comparison the corresponding figures are given for the living white population of Baltimore in 1920.

In the same way it is desirable to examine the age distributions of the dead in the present material. Table XVII gives the percentage distribution of dead persons, and of the deaths of white persons in Baltimore in 1910, for comparison. This is taken as the year for comparison of deaths in the general population, rather than any later year, because it comes nearer to

TABLE XVI

PERCENTAGE DISTRIBUTION IN RESPECT OF AGE OF (*a*) TOTAL LIVING WHITE POPULATION OF BALTIMORE AGED 15 YEARS AND OVER IN 1920, AND (b) LIVING PERSONS IN PRESENT EXPERIENCE

Age	Baltimore		Present Material	
	Males	Females	Males	Females
15–19	11.6	11.9	11.8	10.1
20–24	13.2	13.2	11.7	11.3
25–29	13.3	12.9	9.8	10.1
30–34	11.8	11.2	10.0	9.3
35–39	10.9	10.5	9.2	10.9
40–44	8.9	8.8	8.9	8.8
45–49	8.3	7.9	9.1	8.3
50–54	6.9	7.0	6.9	7.3
55–59	5.1	5.2	5.4	4.4
60–64	4.2	4.4	5.8	4.8
65–69	2.7	3.0	4.2	5.4
70–74	1.6	2.0	3.2	4.1
75–79	0.9	1.2	2.0	2.5
80–84	0.3	0.6	1.2	1.4
85–89	0.1	0.2	0.5	0.5
90–94	0.03	0.05	0.1	0.6
95–99	0.006	0.009	0.2	0.2
100 and over	0.001	0.002	0.07	...

the average absolute date of the deaths in the present material than would any later year.

There are obvious systematic discrepancies between the percentage age distributions of the persons in this experience, and the present white population of Baltimore, both in respect of the living and of the dead. But plainly it is unfair to compare this experience, covering three to five generations, with a present-day general population, because in the last 75 years the age distribution of the living population of Baltimore has changed, chiefly as a result of the great industrial development

TABLE XVII

PERCENTAGE AGE DISTRIBUTION OF DEATHS OF WHITE PERSONS AGED 15 YEARS AND OVER IN BALTIMORE, IN 1910, AND OF DEAD PERSONS IN THE PRESENT MATERIAL

Age	Baltimore, 1910		Present Material	
	Males	Females	Males	Females
15–19	3.0	3.3	1.9	2.5
20–24	4.3	5.1	2.9	4.8
25–29	5.4	4.9	5.5	5.5
30–34	5.7	5.2	7.4	5.8
35–39	7.5	5.2	7.8	6.8
40–44	6.8	5.6	8.6	6.0
45–49	7.4	6.5	7.2	5.3
50–54	8.8	7.1	9.6	8.8
55–59	9.8	8.1	7.4	7.0
60–64	10.8	8.7	7.0	7.9
65–69	10.3	9.5	8.5	10.3
70–74	8.0	10.3	9.2	8.8
75–79	6.4	9.3	7.0	9.5
80–84	3.4	6.0	4.9	4.8
85–89	1.5	3.7	2.6	4.0
90–94	0.7	1.2	1.8	1.6
95–99	0.07	0.2	0.7	0.6
100 and over	0.03	0.07	0.1	...

of the city. So then it is necessary to get at once another basis of comparison.

Considering the shortcomings of official statistics the best it has been possible to do is to determine for the entire population of Baltimore, taking both sexes and whites and negroes together, the average percentage distributions for the census years 1830 to 1920 inclusive. The data for such computation are taken from Howard's (78) Tables 10 and 136. This period of time is roughly the same as that covered by the present experience. The distributions are shown in Table XVIII.

The nature of the discrepancies between the present material

TABLE XVIII

COMPARISON BETWEEN THE AVERAGE PERCENTAGE AGE DISTRIBUTION OF LIVING POPULATION AND OF DEATHS FOR (a) THE TOTAL POPULATION OF BALTIMORE AT THE CENSUS YEARS FROM 1830 TO 1920 INCLUSIVE, AND (b) THE TOTAL PRESENT MATERIAL

Age group	Living		Dead	
	General population of Baltimore	Present Family History material	General population of Baltimore	Present Family History material
20–29	33.6%	24.1%	15.6%	9.3%
30–39	25.9	22.1	16.1	14.6
40–49	18.6	19.7	15.6	14.5
50–59	11.9	13.5	15.9	16.9
60–69	6.5	11.4	17.0	16.8
70–79	2.7	6.5	12.8	17.3
80 and over	0.7	2.6	7.0	10.7

and the general population, averaged over a comparable period of time, is clearly shown in Figs. 6 and 7.

The differences between the present material and the general population are made plain by these diagrams. In respect of both living and dead persons, the present material is in defect in the earlier years of life, and in excess in the later years.

The reason for these discrepancies has been given much thought and careful study. I have concluded, as the result of thorough consideration of many different aspects of the material, that these discrepancies do not mean, as might at first be thought, that the present data are unreliable, or a bad random sample of the working-class population. The differences arise, I believe, because (a) the material is composed entirely of statistics in which the unit of collection is the *family*, and not as in census or registration returns, the individual;

and (b) family statistics in the nature of the case necessarily differ in age composition from general population statistics. The chief points of this necessary difference are two in number. They are:

(1) Family statistics tend always to show a deficiency of young persons, both living and dead, as compared with the

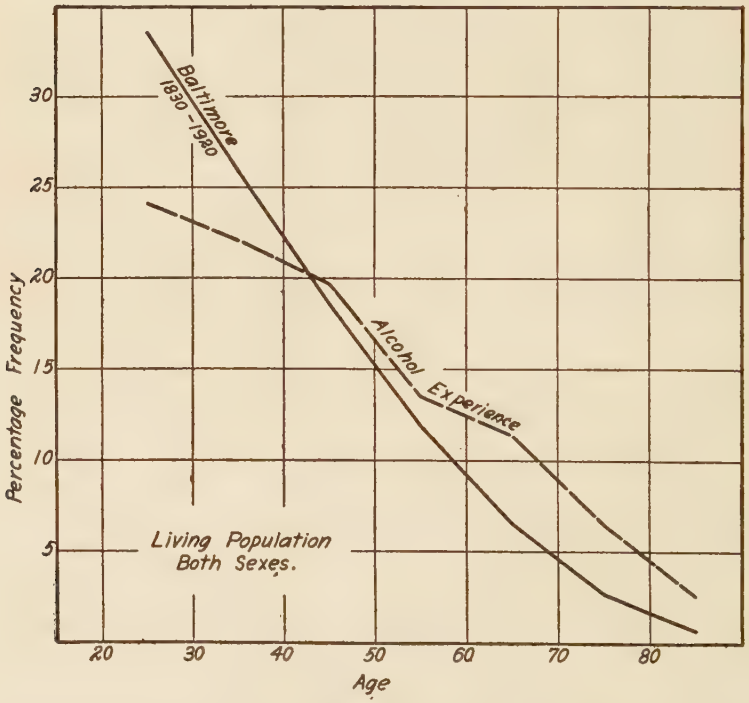

FIG. 6. The percentage age distribution of (a) the total living population (both sexes and all races) of Baltimore, averaged for the census years 1830 to 1920 inclusive (solid line), and (b) the living persons at the time of record in the present Family History material, averaged for the two sexes (broken line).

general population of large urban communities, because no young persons unattached to relatives (parents, etc.) ever ap-

pear in the family statistics. On the other hand, a great many such persons appear in the general population statistics of a city, because they migrate in to work in factories, shops, etc., leaving their families behind in the country or in other cities.

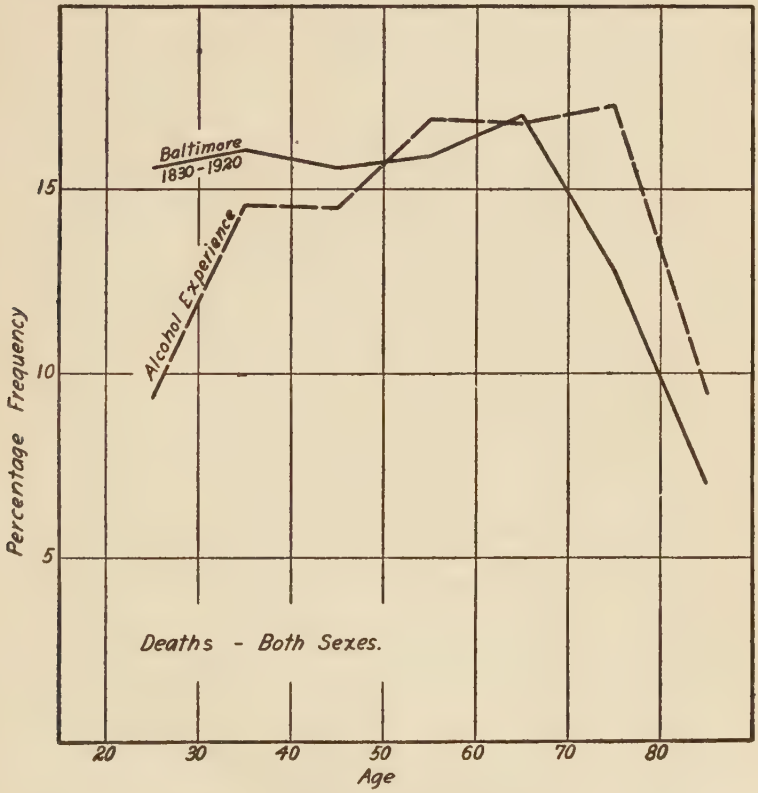

FIG. 7. Like Fig. 6, but for deaths.

(2) Family statistics tend to show an excess of old persons, both living and dead, in comparison with general population statistics, because they include an excess of *parents*, and parents, as a class, live longer than the average duration of

life of the whole population. This fact is shown in Table XIX taken from Pearl (6).

TABLE XIX

MEAN AGE AT DEATH OF PARENTS

	Mean age of father at death	Mean age of mother at death	Authority and comments
English Quakers	68.37 ± 0.31	67.95 ± 0.33	Beeton and Pearson (Biometrika 1 : 60, 1901): parents of adult sons.
English Quakers	69.55 ± 0.27	68.70 ± 0.30	Beeton and Pearson: parents of adult daughters.
Baltimore working class[3]	69.83 ± 0.97	67.52 ± 0.83	Pearl (Am. J. Hyg. 3:78, 84 (Jan.) 1923): parents of non-cancerous persons.[1]
Baltimore working class[3]	63.51 ± 0.48	64.72 ± 0.52	Pearl: parents of non-tuberculous persons.[2]
Whitney family	68.61 ± 0.72	.....	Unpublished data in this laboratory.

[1] Having the same age distribution as a group of persons dying of cancer.
[2] Having the same age distribution as a group of persons dying of tuberculosis.
[3] The "Baltimore working class" group comes from the Family Records from which the present material relative to alcohol is derived.

The two peculiarities of family statistics which have been mentioned appear to be entirely sufficient to account for the differences of the present material from the general population in respect of age distribution. The effect of these differences upon the actuarial constants will be discussed farther on.

Obviously if this excess of old and defect of young persons, shown to exist in the present data, were in any degree unequally distributed amongst the several drinking classes, any conclusions drawn regarding life duration in those classes would be under deservedly serious suspicion of being invalid. Is the probability greater that a young person will have been

included in these statistics if he was an abstainer, for example, than if he was a heavy (or a moderate) drinker? Is there a greater probability that an old person will have been included if he was a drinker (heavy or moderate) than if he was an abstainer?

Clearly these, or any similar questions, cannot be answered by the simple process of comparing the age distributions of living and dead in the several drinking categories. Such a comparison is futile. If alcohol has any influence upon mortality, which is precisely the thing we are concerned to find out, this fact will of itself affect the age distributions, and it will be impossible to say what part of any observed differences are due to alcohol, and what part to something else.

The inherent nature of the material suggests that the answer to the questions propounded above is in the negative in both cases, because the unit of collection of the statistics is the *family*. Defining a family for the purpose of this discussion as a father and a mother and their children, every person included in such kinship groups is entered in the basic records from which the present statistics are drawn, regardless of what his or her drinking habits may have been. But, as already explained, it was impossible in practice to get information as to alcohol habits about all individuals. This leads us then to the *really* crucial question: Has the "getting of information" been differential in respect of age between the several drinking groups?

In order to answer this question Table XX has been prepared. This gives the percentage age distribution of all living and dead persons in the first 18 Family Histories in the collection. In this table the material is divided into two fundamental categories, namely (a) those persons about whose alcoholic habits no information was obtained, and (b) those persons about whose alcohol habits information was obtained. The

category (a) therefore comprises those persons whose records do not appear in the statistics of this book, while (b) includes the persons whose records are in the present material. The table includes the case histories numbered 1–5 and 7–18 inclusive, Case No. 6 being omitted because it concerns one of the two negro families tried out as a test case early in the Family History work, to determine whether negroes should be included in the study. These histories are a random sample of the entire material. They were taken as the basis of the test merely because the requisite data had been extracted from them for another purpose, and therefore the construction of Table XX was much less laborious than it would have been with an equally large random sample taken from some other part of the series.

TABLE XX

PERCENTAGE AGE DISTRIBUTIONS, BY SEX AND VITAL STATUS, OF (a) PERSONS FOR WHOM THERE IS NO INFORMATION AS TO ALCOHOL, AND (b) PERSONS FOR WHOM THERE IS SUCH INFORMATION. BOTH (a) AND (b) ARE TAKEN FROM THE SAME RANDOM SAMPLE OF CASE HISTORIES AND TOGETHER INCLUDE ALL PERSONS IN THOSE HISTORIES

Age	(a) Persons for whom there is no information as to alcohol				(b) Persons for whom there is information as to alcohol			
	Living		Dead		Living		Dead	
	Males %	Females %	Males %	Females %	Males %	Females %	Males %	Females %
20–29	28.3	31.8	12.1	14.4	28.1	24.0	9.4	9.7
30–39	30.4	25.5	13.8	18.9	28.1	36.0	15.6	16.1
40–49	18.0	17.4	17.9	15.6	18.7	14.0	21.9	16.1
50–59	9.7	12.5	15.4	12.2	3.1	12.0	6.3	9.7
60–69	7.4	7.5	16.3	16.7	18.7	10.0	21.9	16.1
70–79	4.4	2.5	13.0	12.2	3.1	2.0	18.7	19.4
80–89	1.5	2.8	8.1	6.7	...	...	3.1	6.5
90–99	0.3	...	3.3	3.3	...	2.0	3.1	6.5
Totals	100.0	100.0	99.9	100.0	99.8	100.0	100.0	100.1

The distributions of Table XX are shown graphically in Figs. 8 and 9.

It is evident from both the table and the diagrams that there is no significant difference between the (a) and the (b) series,

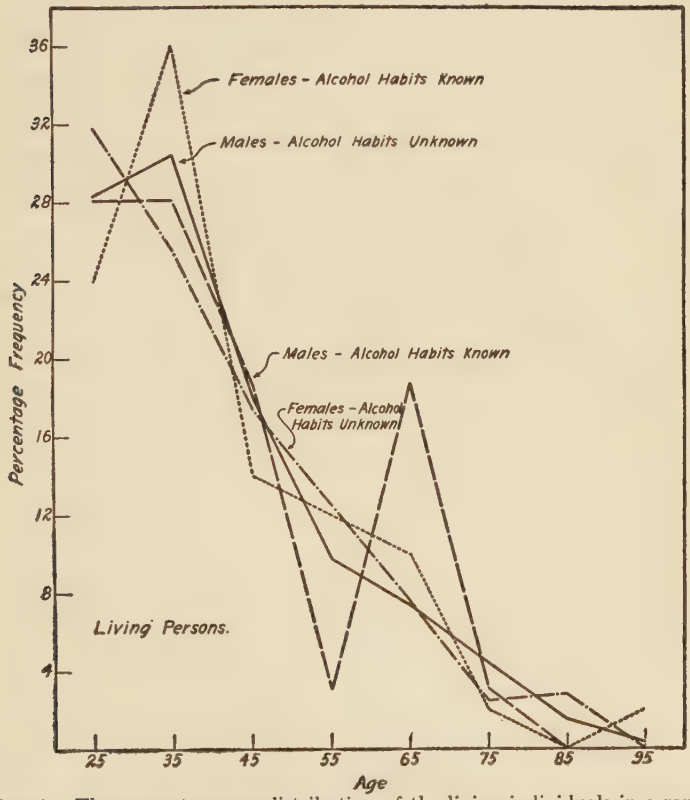

FIG. 8. The percentage age distribution of the living individuals in a random sample, divided according to whether information *was* (males, dash line; females, dot line), or *was not* (males, solid line; females, dash-dot line) obtained as to alcoholic habits.

in respect of the age distribution of the material. Having regard to the absolute size of the samples (1018 individuals in

total, 873 a's and 145 b's) the lines wind in and about each other in such a manner as to indicate that the group for which information was obtained as to drinking habits (which group, of course, includes abstainers as well as drinkers) is a fair random sample, in respect of age distribution, of the Family History material as a whole. Such discrepancies as exist be-

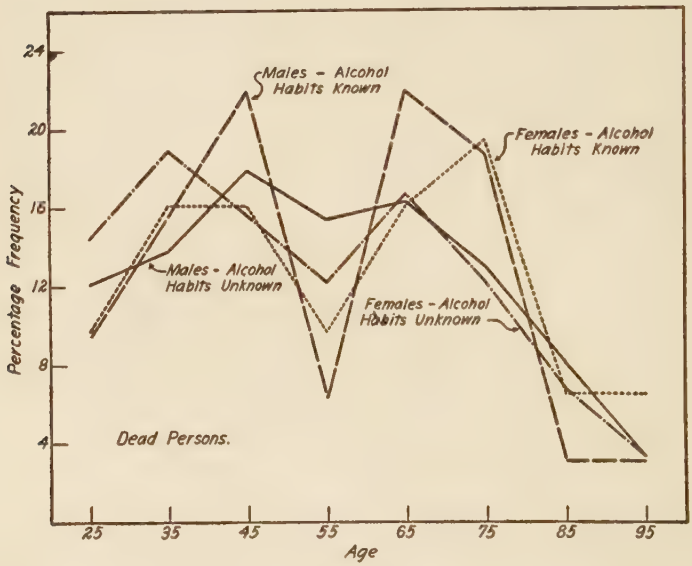

FIG. 9. Like Fig. 8, but for the dead individuals.

tween the present material and the general population, in respect of ages of the living and dead, may be taken to be due, therefore, to the inherent peculiarities of family statistics already described, and not to inadequate or biased sampling.

Turning now to the question of the actuarial methods used, it should be stated first of all that in the life tables for the several drinking categories presented in this book, those per-

sons *whose habits changed* during life, as discussed in Chapter III *supra, have not been included* in the calculations. Their exclusion removes an insignificant fraction only of the total material, and prevents any criticism on the ground that they have been unfairly classified into single drinking categories. All other persons except these "changed habit" individuals are included in the life tables.

I deliberately wish here again to repeat, for the sake of emphasis, what was said at the beginning of this chapter, namely that all of the life tables in this book are based upon, and include, *both* those persons living at the time of record and those persons who had died prior to the time of record. The present material is, therefore, from an actuarial point of view, precisely comparable with, and essentially the same as, the general experience of a life insurance company. If such a company is ever justified in calculating a life table from its own experience with its own policyholders, living and dead (and I am not aware that anyone has ever denied the validity of such procedure), then it is justifiable to calculate life tables from the experience here under review. In one respect the present material is actuarially better than that of any insurance company, because here there is no necessity to correct for "lapses of policies" (that is, withdrawal from the exposed to risk group by reason of failure to pay premiums). Here a person goes out of the exposed to risk group only because of death.

In the actuarial computations the central death rates are based on a five-year grouping of the raw data, the groups being centered at 20, 25, etc., years to avoid any disturbing influence of the usual tendency to concentrate on multiples of five in the statement of age. The years of exposure to risk in each quinquennial period were obtained as follows:

5 (sum of living and dead for all years after end of period).
+ 4.5 (sum of persons living and dying in last year of period).
+ 3.5 (sum of persons living and dying in fourth year of period).
+ 2.5 (sum of persons living and dying in third year of period).
+ 1.5 (sum of persons living and dying in second year of period).
+ 0.5 (sum of persons living and dying in first year of period).

The total deaths in each period divided by the years of exposure to risk in the same period give the m_x values, the central death rates, from which q_x's (the life table death rates) were obtained by the usual formula:

$$q_x = \frac{2m_x}{2 + m_x}$$

The q_x values were graduated by standard curve fitting procedures customarily used in this type of work. From the q_x values were computed in the usual way the other important life table functions l_x and $\mathring{e}_x$. A discussion of the mathematical methods used in graduating the raw values in the life tables presented in this book is given in Appendix I. Being wholly of technical interest it seems proper to make this disposition of it rather than to encumber the text with it. The definitive life tables were computed as of 100,000 persons entering the experience at age 30. The q_x's were actually computed from age 20 on. In the nature of the case, however, it would be unfair to the abstainers to start the tables at age 20. The reason is that the deaths prior to about age 30 include a number of per-

sons who were actually abstainers up to the time they died, but who, if they had lived, would have become drinkers and passed out of the abstainer into one or another of the drinking classes. The result is to make the abstainers' death rates spuriously high at the early ages. But a careful analysis of the present material, backed by common-sense experience on the point, shows that by the time age 30 is reached substantially all persons who are ever going to leave the total abstainer group and pass into a drinking group have already done so. From age 30 on all groups are justly comparable in respect of their mortality. The magnitude of the experience, in terms of person-years exposed to risk, upon which the definitive life tables of Chapter V are based, will be shown in connection with each such table.

The reader not familiar with the terminology of actuarial work, or with the meaning of life tables, will do well to consult some elementary discussion of this matter, such as that of the present writer (66) or Henderson (77). The best available detailed discussion of the theory of life table computation is that of Glover (74).

Complete life tables are presented in Appendix II for the following groups:

1. Abstainers — Male.
2. Abstainers — Female.
3. All moderate males (moderate occasional + moderate steady + moderate, unspecified as to frequency).
4. All moderate females (moderate occasional + moderate steady + moderate, unspecified as to frequency).
5. All heavy males (heavy occasional, otherwise abstainer + heavy occasional, otherwise moderate + heavy steady + heavy, unspecified as to frequency).

There are too few heavy drinking females in the experience to warrant the construction of a life table for them.

Short tables have been constructed for certain of the smaller groups, and their results will be presented in the text of Chapter V.

The Mortality Experience of the Group as a Whole

It has been alleged by some critics of the preliminary papers which have been published regarding this investigation, that the material is not normal or representative. It has been said, in effect, that the 5248 persons in this experience were individually abnormal, and abnormal in such a manner as to lead to wholly wrong conclusions as to the influence of alcohol upon mortality. The only reason appearing in support of the allegation of abnormality, has been that the results were not in accord with the expectations, or the desires of the critics. In short, because the preliminary results of the inquiry ran counter to the prejudices of some people, it is charged that the material is abnormal, and that therefore the results are to be dismissed as worthless.

In Chapters II and III a careful detailed description has been given of racial, social and economic characteristics of the group, and it was shown that in these respects there is nothing which can be regarded as abnormal about the material. It is, in so far, a random sample of the working-class population of Baltimore. In the present chapter it has been shown that the age distributions of the living and the dead in this material differ from those of the general white population of Baltimore, only in such manner and degree as is to be expected from the fact that these are *family* statistics. It now remains to examine the actuarial characteristics of the group as a whole. Are the characteristics of this group in respect to the age distribution of its mortality alike or different from those of the general population, and if so by how much?

It will be appropriate to exhibit first the magnitude of the experience in actuarial units. This is done in Table XXI.

TABLE XXI

PERSON-YEARS EXPOSURE TO RISK IN THE PRESENT MATERIAL AS A WHOLE

Central age	Person-years exposure to risk of *males*, in the 5 year age class of which the central age is that given	Person-years exposure to risk of *females*, in the 5 year age class of which the central age is that given
20	14,196.5	9,961.5
25	13,044.5	9,003.0
30	11,819.5	8,079.5
35	10,519.0	7,216.0
40	9,198.0	6,251.0
45	7,836.5	5,404.5
50	6,595.5	4,595.5
55	5,401.5	3,814.0
60	4,351.0	3,191.0
65	3,387.0	2,517.5
70	2,439.0	1,789.5
75	1,477.0	1,128.0
80	860.5	611.0
85	446.5	311.0
90	191.5	115.5
95	76.5	36.5
100	13.5	4.0
Total	91,853.5	64,029.0

Clearly an experience which includes over 155,000 person-years exposure cannot be lightly dismissed as too small to base any conclusions upon.

For the material as a whole, without distinction as to the several classes in respect of alcohol consumption, a life table has been computed. It will not be necessary to present this table in detail, because its only purpose is to enable a general

comparison of the actuarial characteristics of this material with other life tables. It will suffice for this purpose to present only one of the life table functions. For this purpose the expectation of life has been chosen. The data are given in Table XXII. For comparison the expectation of life at the same ages for the total white population of Baltimore in 1919–1920 is inserted from Foudray's (48) tables.

TABLE XXII

Complete Expectation of Life in Years, for the Total Present Material Compared with the White Population of Baltimore, 1919–20

Age	Males		Females	
	Alcohol	Baltimore	Alcohol	Baltimore
22	40.75	40.64	45.02	42.72
27	36.69	36.67	41.04	39.03
32	32.99	32.82	37.20	35.28
37	29.54	29.03	33.45	31.41
42	26.32	25.30	29.80	27.53
47	23.28	21.76	26.26	23.81
52	20.42	18.36	22.85	20.13
57	17.74	15.18	19.58	16.75
62	15.22	12.27	16.49	13.62
67	12.87	9.73	13.60	10.62
72	10.71	7.69	10.95	8.25
77	8.73	5.93	8.56	6.48
82	6.92	4.26	6.46	4.81
87	5.33	2.78	4.74	3.08
92	3.87	1.50	3.48	1.73

From the table it is seen that, so far from the present material being in any respect a substandard group, in the actuarial sense, it actually exhibits, in both sexes, a higher expectation of life at all ages included in the table than does the general white population of Baltimore. The amount of the excess expectation in the case of the males is insignificant in the earlier

years, but increases relatively in the later years of the life span. In the case of the females the excess is quite considerable throughout.

A significant excess in the expectation of life at advanced ages in this material is precisely what would be expected from the facts brought out earlier in this chapter, consequent upon the circumstance that the basis of the present material is *family* statistics. In fact a life table is only another way of expressing these same facts, an excess of living persons at advanced ages caused by a relative preponderance of parents in statistics for which the family is the unit of collection. But the general trend of the expectation of life in the present material corresponds reasonably to what is found in the Baltimore white population, or to other general populations. The only essential particular in which this material deviates from the usual actuarial experience with general urban populations, is in its higher absolute expectations at advanced ages, and the reason for this discrepancy has been shown to be the family basis of the statistics. This fact will be still further demonstrated in the next chapter.

In view of these circumstances it is possible to proceed with considerable confidence to the comparison of the several drinking classes in respect of their mortality. The only caution necessary is to remember that all the death rates beyond roughly age 50 are absolutely lower (or the expectation of life higher) than those which would be shown by a random sample of a general population. The comparisons of one drinking class with another will, however, be in no way affected by this circumstance, and it is in these comparisons that the chief interest of the investigation lies.

CHAPTER V

THE MORTALITY EXPERIENCE OF DIFFERENT DRINKING CLASSES COMPARED

We come now to the central problem of this study, for the discussion of which all that has preceded has been simply preparatory. The question at issue may be precisely put in this manner: In what direction and to what degree does the duration of life of moderate drinkers, as a group, differ from the duration of life of total abstainers, as a group, or of heavy drinkers, as a group? Methodologically the proper way to proceed to get an answer to this question is to construct life tables, by standard actuarial methods, for groups of abstainers, of moderate drinkers, and of heavy drinkers. As explained in earlier chapters this is precisely what has been done in this investigation.

It is the purpose of the present chapter to exhibit the results of this procedure. The detailed life tables are given in Appendix II. Here in the text only such digests of them will be presented as are essential to a clear understanding of the conclusions to which they lead.

There is an extensive literature on the influence of alcohol upon mortality, but the discussion of this may most profitably be deferred until the results of the present study are before the reader. Then the whole *corpus* of present evidence on the problem can be dealt with as a unit.

The first comparison may well be between the three drinking categories, taking all the material together; that is, comparing abstainers with all moderates (moderate occasionals + moderate steadies + moderate unspecified as to frequency), and with

TABLE XXIII

EXPECTATION OF LIFE, IN YEARS, IN THE SPECIFIED GROUPS OF MALES

Age x	Abstainer	All moderate	All heavy	Difference in favor of moderates as compared with abstainers	Difference in favor of moderates as compared with heavies	Difference in favor of abstainers as compared with heavies	White males in cities of O.R.S. 1901 (Glover's Table 27)
30	36.34	36.75	28.57	+0.41	+8.18	+7.77	31.89
35	32.69	33.22	26.31	+ .53	+7.91	+7.38	28.44
40	29.06	29.69	22.41	+ .63	+7.28	+6.65	25.06
45	25.47	26.21	19.85	+ .74	+6.36	+5.62	21.78
50	21.99	22.84	17.57	+ .85	+5.27	+4.42	18.56
55	18.64	19.60	15.51	+ .96	+4.09	+3.13	15.51
60	15.49	16.56	13.61	+1.07	+2.95	+1.88	12.80
65	12.58	13.75	11.83	+1.17	+1.92	+ .75	10.32
70	9.97	11.21	10.13	+1.24	+1.08	− .16[1]	8.20
75	7.71	8.96	8.45	+1.25	+ .51	− .74	6.31
80	5.90	7.01	6.78	+1.11	+ .23	− .88	4.78
85	4.55	5.35	5.18	+ .80	+ .17	− .63	3.60
90	3.44	3.99	3.88	+ .55	+ .11	− .44	2.68
95	2.54	2.90	2.83	+ .36	+ .07	− .29	1.97
Person-years exposed to risk in the group	21,594.0	43,011.5	24,371.0	..	..	..	..

[1] The differences in this column, from this point on to the bottom, are in favor of the heavy drinking group as compared with the abstainers.

all heavy drinkers (heavy occasional, otherwise abstainers + heavy occasional, otherwise moderate + heavy steady + heavy unspecified as to frequency). The life table function chosen for the first succinct presentation of the results here in the text is the expectation of life; that is, the mean after-life-time of persons living at age x. Table XXIII gives this

TABLE XXIV

EXPECTATION OF LIFE, IN YEARS, IN THE SPECIFIED GROUPS OF FEMALES

Age x	Abstainers	All moderate	Difference in favor of moderates as compared with abstainers	White females in cities of O.R.S., 1901 (Glover's Table 29)
30	37.63	40.95	+3.32	34.45
35	33.91	36.97	+3.06	30.88
40	30.18	32.97	+2.79	27.30
45	26.50	29.03	+2.53	23.78
50	22.92	25.18	+2.26	20.28
55	19.50	21.50	+2.00	17.03
60	16.30	18.04	+1.74	14.05
65	13.36	14.85	+1.49	11.34
70	10.74	11.99	+1.25	8.97
75	8.47	9.50	+1.03	6.95
80	6.57	7.38	+ .81	5.25
85	5.04	5.62	+ .58	3.99
90	3.77	4.18	+ .41	3.02
95	2.76	3.02	+ .26	2.25
Person-years exposed to risk in the group	41,449.0	20,900.00	..	..

function at quinquennial years from 30 to 95, together with differences for comparison, for the males in the present experience. Table XXIV gives the same data in the same way for the females in this experience. The female table differs from that of the males in the omission of the columns relating to heavy drinkers. The experience of this category of females is too small to warrant any attempt to calculate expectations of life. Later on the significance of what material there is for heavy drinking females will be discussed.

In both Tables XXIII and XXIV there is added at the end

a column giving the corresponding value from Glover's (74) life table for white persons (male or female) in cities of the Original Registration States in 1901.

The first point which strikes one in examining Tables XXIII and XXIV is that, from age 30 on, the expectation of life of the moderate drinkers, whether male or female, is *higher* at all ages than the expectation of life of abstainers. In the males the differences in favor of the moderates are probably for the most part, or perhaps entirely, too small to be regarded as statistically significant, having regard to the errors incident to sampling. In the females the differences between abstainers and moderate drinkers up to ages 50 or 55, may perhaps be regarded as statistically significant. But, in any case, *Tables XXIII and XXIV certainly give no evidence that the expectation of life from age 30 on is in any degree impaired by the moderate consumption of alcoholic beverages, as defined in this book.*

The relation between the moderate drinkers, the heavy drinkers, and the abstainers, in respect of duration of life, is shown graphically in Fig. 10 (for males) and Fig. 11 (for females).

The amount of the excess, in mean after-life-time, of the moderate drinkers over the abstainers, begins for the males at a value of 0.41 year at age 30, and gradually increases with advancing age to a maximum of 1.25 years at age 75. After that age the difference drops again and amounts to only 0.36 year at age 95. In the females, the expectation of life at age 30 of the moderate drinkers is 3.32 years greater than that of the abstainers. The difference between the two groups of women in mean after-life-time becomes steadily smaller with advancing age, reaching the value of 0.26 year at age 95.

The heavy drinking males have an expectation of life markedly inferior to that experienced in both the other groups,

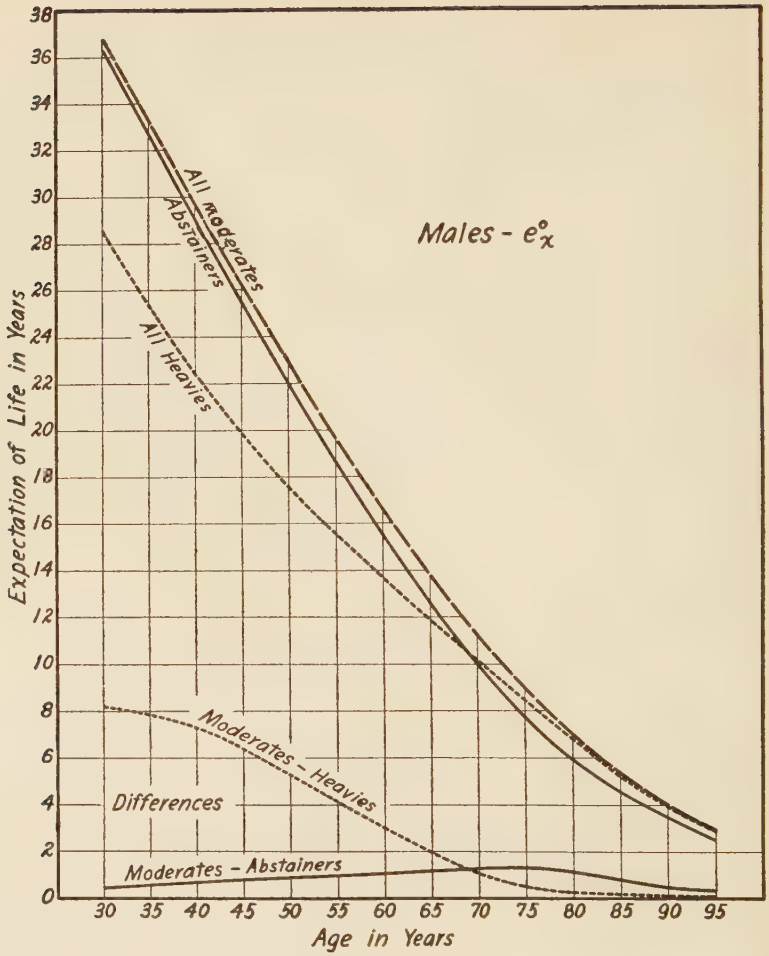

FIG. 10. The expectation of life (e°_{x}) in years, for males falling in the three drinking categories (a) abstainers (solid line), (b) moderate drinkers (dash line), (c) heavy drinkers (dot line). At the bottom of the diagram are plotted the differences in years (a) between moderate drinkers and abstainers (solid line), and (b) between moderate and heavy drinkers. Data from Table XXIII.

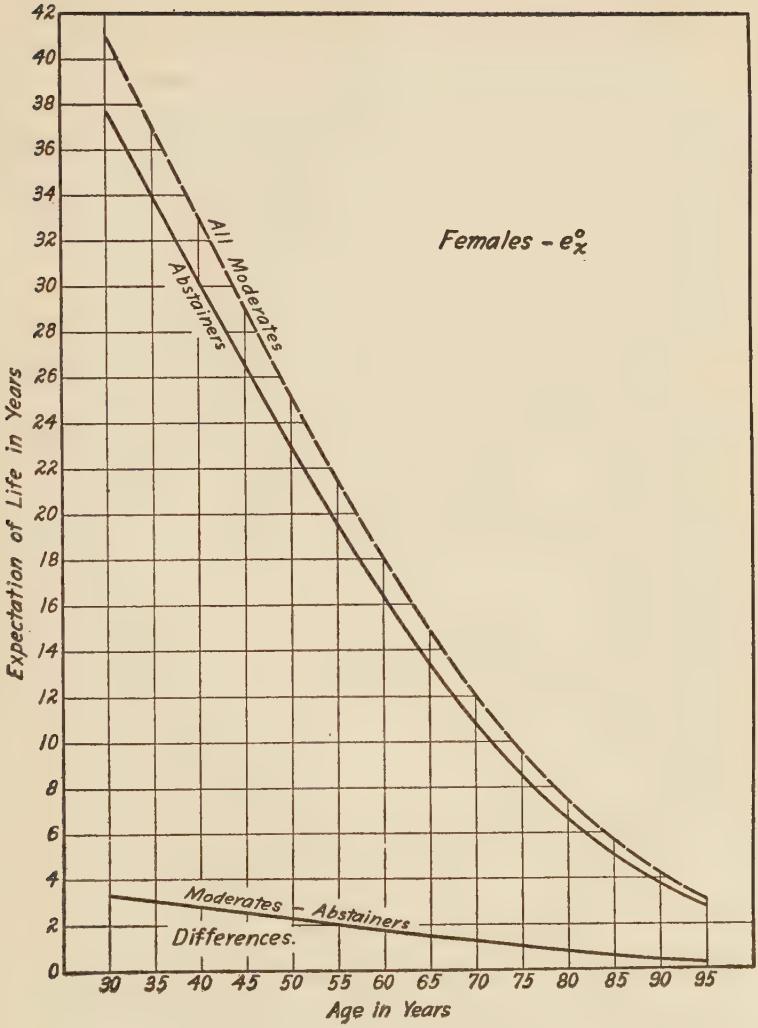

FIG. 11. The expectation of life (e_x°), in years, for females falling in the two categories (a) abstainers (solid line), and moderate drinkers (dash line). At the bottom of the diagram are plotted the differences, in years, between moderate drinkers and abstainers (solid line). Data from Table XXIV.

at the earlier ages. The greatest differences are between the moderate and the heavy drinkers. At age 30 the moderate drinkers have an expectation of life 8.18 years greater than do the heavy drinkers. This difference diminishes with advancing age, at first slowly, then more rapidly, and finally slowly again. At age 75 the expectation of life of heavy drinkers is only 0.51 of a year poorer than that of the moderate drinkers, and at age 90 only 0.11 year. At age 69 the abstainer males and the heavy drinking males in this material had precisely the same expectation of life, namely 10.46 years. From that age on to the upper end of the life span, the life tables indicate a somewhat superior expectation of life for the heavy drinkers as compared with the abstainer. But the differences are absolutely small, arising to a maximum of only 0.88 year at age 80.

The explanation of this seemingly paradoxical result that at advanced ages the mean after-life-time of heavy drinkers is quite as good as that of abstainers, or even a little better, probably is that the excess mortality from heavy drinking in early life is selective in considerable degree. On this view the heavy drinker who survives to age 70 has proved himself to be a tough customer, not easily killed. He is presumably a sounder person than is his abstaining brother who reaches the age of 70. For while the latter has had to withstand successfully and survive only the ordinary environmental vicissitudes of life, the former has not only had to do that much, but also to survive the effects of deleterious quantities of alcohol. Therefore while fewer of a cohort of 1000 heavy drinkers, starting together at age 30, reach the age of 70 than attain that age from a similar cohort of abstainers, those who do are presumably superior persons constitutionally. The findings of Heron in his study of female inebriates, which are described in Chapter I, are of interest in this connection. Another point to be considered is that some extremely old, consistently heavy drinkers

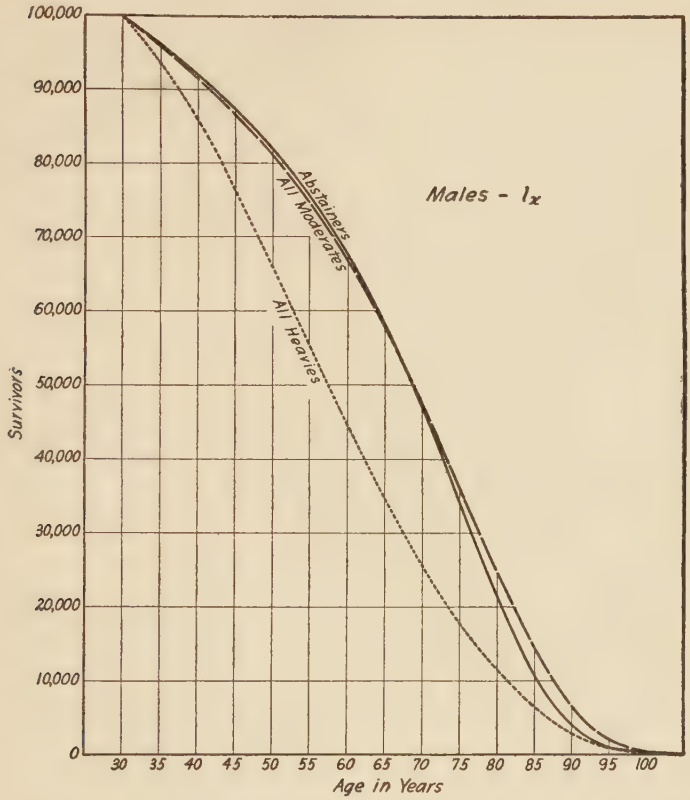

FIG. 12. The number of surviving males out of 100,000 starting together at age 30, in the three drinking categories: (a) abstainers (solid line), (b) moderate drinkers (dash line), and (c) heavy drinkers (dot line).

are known to be "refractory" to alcohol. Follet (219) has discussed this point. He describes a man, living and well at age 85, who had taken large amounts of alcohol daily since the age of 16. He was accustomed to get drunk twice a week. Follet believes that the basis of such resistance to the ravages of alcohol is to be found in an unusually sound inherited constitution.

The expectation of life $\mathring{e}_x$ is, in a sense, the most highly derivative of all the functions of a life table. In particular the values of this function are somewhat heavily affected by the

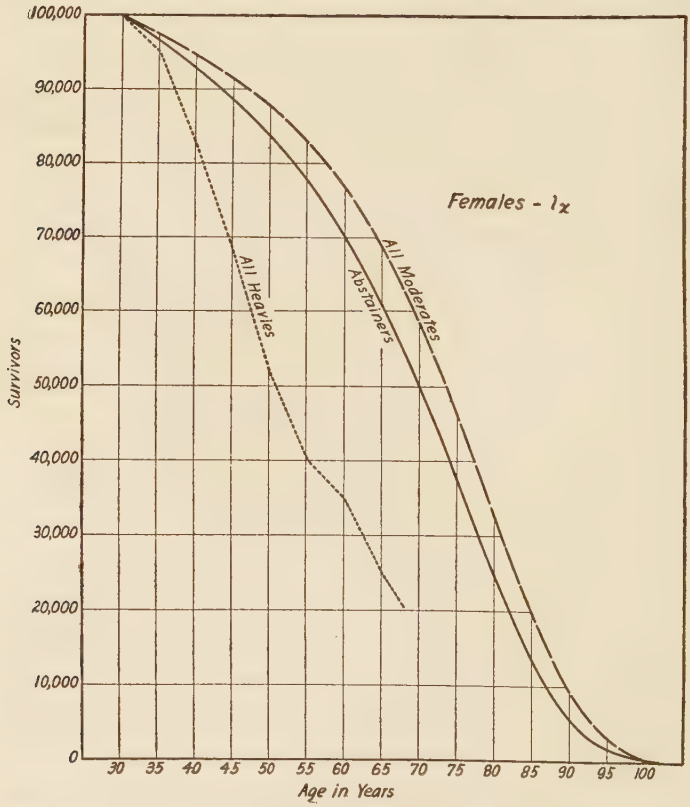

FIG. 13. The number of surviving females out of 100,000 starting together at age 30, in the three drinking categories: (a) abstainers (solid line), (b) moderate drinkers (dash line), and (c) heavy drinkers (dot line).

always meager, and to that extent unreliable, data at the most advanced ages near the upper end of the life span. On this account it will be well to examine critically some of the other

functions of these alcohol life tables before reaching final conclusions upon so important a matter. Let us therefore next consider the survivorship function l_x. This may be done graphically. To this end Figs. 12 and 13 are presented. They are the graphs of the values shown in Table XXV.

TABLE XXV

NUMBER OF SURVIVORS OUT OF 100,000 STARTING TOGETHER AT AGE 30, IN THE SPECIFIED GROUPS

Age x	Males				Females		
	Abstainers	All moderate	All heavy	White males in cities of O.R.S., 1901. Glover's Table 27	Abstainers	All moderate	White females in cities of O.R.S., 1901. Glover's Table 29
30	100,000	100,000	100,000	100,000	100,000	100,000	100,000
35	96,152	95,878	93,719	95,010	96,469	97,423	95,714
40	91,995	91,496	85,767	89,415	92,715	94,662	91,156
45	87,328	86,642	76,391	83,050	88,491	91,493	86,013
50	81,914	81,084	66,034	76,028	83,526	87,660	80,325
55	75,470	74,579	55,253	67,744	77,499	82,832	73,112
60	67,686	66,887	44,616	57,548	70,065	76,609	64,127
65	58,262	57,835	34,619	46,230	60,911	68,562	53,502
70	47,060	47,428	25,631	33,744	49,926	58,368	41,168
75	34,375	36,025	17,891	21,902	37,466	46,102	28,205
80	21,332	24,504	11,545	11,594	24,654	32,613	16,271
85	10,518	14,169	6,484	4,580	13,475	19,691	7,137
90	3,913	6,420	2,832	1,215	5,691	9,423	2,219
95	965	2,018	851	182	1,639	3,170	430

The close similarity between the life curves for the abstainers and moderate drinking males is again clearly brought out in Fig. 12. Up to age 70 there is only a slight difference between the two curves. From that point on to near the end of the life span the two curves diverge, that for the moderate drinkers

lying above that for the abstainers, indicating a greater survival at those ages for the former group. Again there is no evidence that moderate drinking as defined in this study, in any significant degree impairs the survival of males practising it, as compared with total abstainers.

At all ages from 30 on the survivorship line for heavy drinkers lies well below that of either abstainers or moderate drinkers. At a point between 58 and 59 years of age one-half of the original 100,000 heavy drinkers starting together at age 30, have passed away. But it is not until 10 years later (between 68 and 69) that one-half of the abstainers and the moderate drinkers have died off. *Clearly heavy drinking, as defined in this book, definitely and seriously impairs life duration in males.*

Turning to the female curves (Fig. 13) the chief difference to be noted is that the survivorship curve for moderate drinkers lies above that for abstainers at all ages from 30 on, indicating again that moderate drinking, as defined in this study, has had no deleterious effect upon life duration. On the contrary there are surviving at age 70, out of the original groups of 100,000 each starting at age 30, 16.9 per cent more moderate drinking females than abstaining females.

There are plotted in Fig. 13 (as a dotted line) the raw, unsmoothed figures for the survivorship of the few heavy drinking females. The experience is too small to warrant graduation, as has already been pointed out, but the rough values, so far as they go, unmistakably indicate the deleterious effect of heavy drinking, as here defined, in the female just as in the male.

The basic data upon which any life table is constructed are the death rates at ages. Let us therefore next examine with some care the q_x values for these tables. Tables XXVI and XXVII give a summarized statement at quinquennial ages.

These tables are constructed on the same plan as Tables XXIII and XXIV, but for mortality rates (1000 q_x) instead of expectation of life ($\mathring{e}_x$).

TABLE XXVI

LIFE TABLE DEATH RATES (q_x) PER 1000 LIFE YEARS EXPOSURE IN THE SPECIFIED GROUPS OF MALES

Age x	Abstainer	All moderate	All heavy	Difference in favor of moderates as compared with abstainers	Difference in favor of moderates as compared with heavies	Difference in favor of abstainers as compared with heavies	White males in cities of O.R.S., 1901. Glover's Table 27
30	7.54	8.14	11.19	− 0.60[1]	+ 3.05[2]	+ 3.65[3]	9.47
35	8.33	8.85	15.59	− 0.52	+ 6.74	+ 7.26	11.25
40	9.63	10.12	20.67	− 0.49	+ 10.55	+ 11.04	13.54
45	11.62	12.09	26.31	− 0.47	+ 14.22	+ 14.69	16.12
50	14.61	15.01	32.42	− 0.40	+ 17.41	+ 17.81	20.31
55	19.07	19.26	39.02	− 0.19	+ 19.76	+ 19.95	27.68
60	25.79	25.39	46.27	+ 0.40	+ 20.88	+ 20.48	38.15
65	36.03	34.21	54.53	+ 1.82	+ 20.32	+ 18.50	52.45
70	51.85	46.82	64.52	+ 5.03	+ 17.70	+ 12.67	73.08
75	76.65	64.75	77.35	+ 11.90	+ 12.60	+ 0.70	101.90
80	116.10	89.95	94.79	+ 26.15	+ 4.84	− 21.31	146.45
85	157.92	126.89	132.72	+ 31.03	+ 5.83	− 25.20	204.53
90	214.80	179.00	185.83	+ 35.80	+ 6.83	− 28.97	279.06
95	292.17	252.51	260.19	+ 39.66	+ 7.68	− 31.98	374.58

[1] When the moderate group has a *higher* death rate than the abstainer the difference is marked − because it is in favor of the abstainer and against the moderate.

[2] The moderate group has *lower* death rates than the heavy group at all ages, hence the differences are all marked +.

[3] When the death rates for the abstainer group are *lower* than those for the heavy group, the differences are marked +.

The death-rate curves of Tables XXVI and XXVII are shown graphically in Figs. 14 and 15.

These most basic and trustworthy life table constants, the age specific death rates, exhibited in the last two tables and

TABLE XXVII

LIFE TABLE DEATH RATES (q_x) PER 1000 LIFE YEARS EXPOSURE, FOR THE SPECIFIED GROUPS OF FEMALES

Age x	Abstainer	All moderate	Difference in favor of moderates as compared with abstainers	White females in cities of O.R.S., 1901. Glover's Table 29
30	7.00	5.11	+ 1.89[1]	8.30
35	7.52	5.45	+ 2.07	9.27
40	8.62	6.27	+ 2.35	10.72
45	10.44	7.69	+ 2.75	12.50
50	13.28	9.97	+ 3.31	16.30
55	17.59	13.51	+ 4.08	22.42
60	24.05	18.93	+ 5.12	31.35
65	33.71	27.15	+ 6.56	43.60
70	48.02	39.42	+ 8.60	63.13
75	69.01	57.36	+ 11.65	88.85
80	99.24	82.78	+ 16.46	130.93
85	138.04	118.17	+ 19.87	183.99
90	192.02	168.69	+ 23.33	248.09
95	267.10	240.18	+ 26.92	330.76

[1] Signs of the differences as in Table XXVI.

diagrams, show in the clearest possible manner the following results: *In the males the death rates for moderate drinkers are slightly higher than those for abstainers from age 30 to age 55 inclusive, but, in my opinion, there is no statistically significant difference in the specific death rates, in the range of age from 30 to about 70, between abstainers and moderate drinkers, in this experience. In the same range of age the heavy drinking group exhibits a markedly higher rate of mortality than either of the other two groups.*

After age 70 the death rates in an experience of this size begin to lose reliability, just as they do in any life table, but in heightened degree here, because of the meagerness of the

data and their special character as family statistics. On this account great significance cannot be attached to the fact that, at the ages from 75 on the abstainers have higher specific death rates, among the males, than either of the drinking groups.

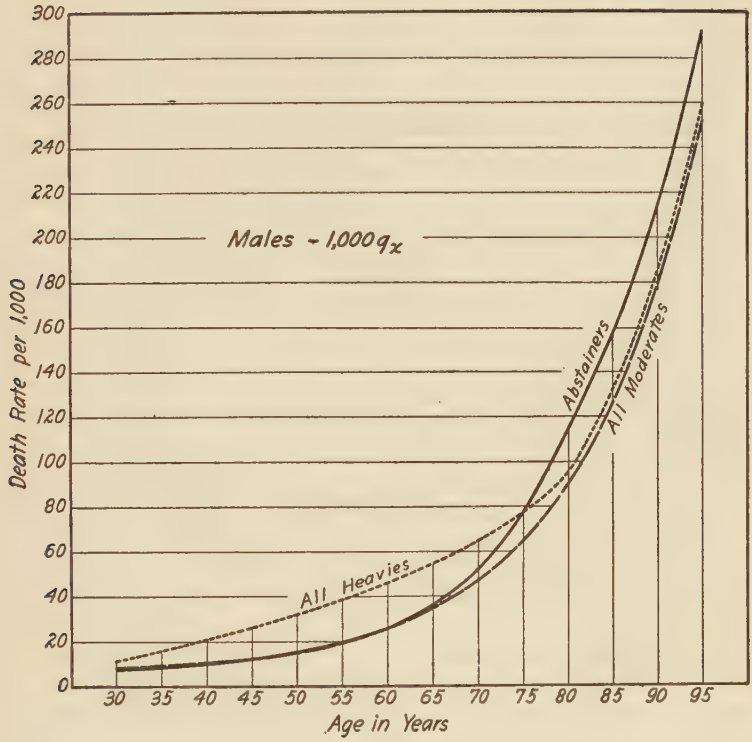

FIG. 14. The life table death rates (1000 q_x) of males in the three drinking categories: (a) abstainer (solid line), (b) moderate drinker (dash line), (c) heavy drinker (dot line).

The result *may* mean what it seems to on its face, but the evidence is not of sufficient magnitude to be in any way conclusive on the point.

In the females the specific death rates at all ages from 30 to the end of the life span are lower in the moderate drinking group than they are in the abstainer group. The differences are relatively considerable at all of these ages.

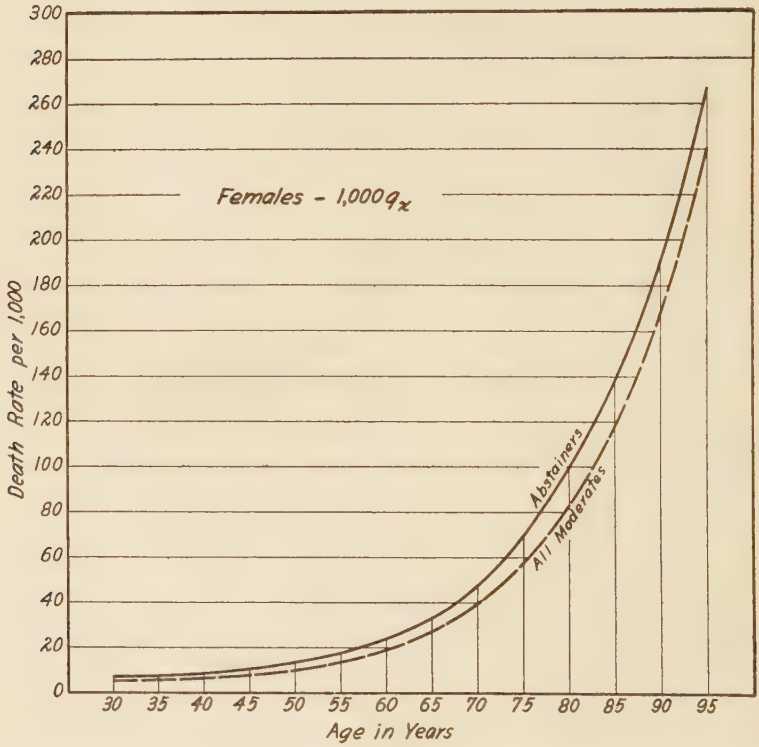

FIG. 15. The life table death rates (1000 q_x) of females in the two drinking categories: (a) abstainer (solid line), (b) moderate drinker (dash line).

Having now seen that the general results of the material as a whole point unequivocally to the conclusion that, in the group of people here studied, the moderate drinking of alcoholic beverages does not impair life expectancy (or increase the

TABLE XXVIII

AGE SPECIFIC DEATH RATES AND EXPECTATION OF LIFE FOR MALES IN THE SPECIFIED DRINKING CATEGORIES

Age x	Moderate occasional		Moderate steady		Heavy occasional		Heavy steady	
	Specific death rate (1000 q_x)	Expectation of life (e_x°)	Specific death rate (1000 q_x)	Expectation of life (e_x°)	Specific death rate (1000 q_x)	Expectation of life ((e_x°)	Specific death rate (1000 q_x)	Expectation of life (e_x°)
32	9.4	34.86	6.2	38.34	7.3	34.32	11.6	27.82
37	10.4	31.47	6.8	34.51	7.9	30.54	16.6	24.60
42	11.9	28.10	8.0	30.69	9.2	26.74	22.2	21.79
47	14.2	24.79	9.8	26.94	11.6	22.98	28.2	19.34
52	17.5	21.59	12.5	23.29	15.6	19.37	34.4	17.18
57	22.2	18.54	16.6	19.80	22.0	15.95	40.9	15.22
62	28.8	15.65	22.8	16.53	32.3	12.81	47.8	13.40
67	38.2	12.98	32.2	13.51	48.9	10.01	55.6	11.64
72	51.4	10.55	46.2	10.80	75.2	7.63	65.0	9.90
77	70.2	8.37	67.5	8.44	116.5	5.70	77.5	8.12
82	96.8	6.46	99.3	6.51	179.5	4.50	95.3	6.27
Person-years exposed to risk in the whole group	12,337.0		19,911.0		3,307.5		9,387.5	

age specific rate of mortality) we may next turn to an examination of the data relative to the different special categories of drinking habits. In Tables XXVIII and XXIX are exhibited the age specific death rates at quinquennial points, and the expectations of life at the same points. In Table XXVIII, for males, these data are given for four groups differing as follows in drinking habits: (a) moderate occasional, (b) moderate steady, (c) heavy occasional, including " heavy occasional,

TABLE XXIX

AGE SPECIFIC DEATH RATES AND EXPECTATION OF LIFE FOR FEMALES IN THE SPECIFIED DRINKING CATEGORIES

Age x	Moderate occasional		Moderate steady	
	Specific death rate ($1000q_x$)	Expectation of life (e_x°)	Specific death rate ($1000\ q_x$)	Expectation of life (e_x°)
32	4.0	37.84	5.9	37.85
37	4.2	33.56	7.1	33.98
42	5.1	29.27	8.6	30.20
47	7.0	25.04	10.8	26.54
52	10.5	20.99	14.1	23.03
57	16.7	17.20	18.1	19.68
62	27.2	13.79	24.2	16.54
67	44.1	10.86	33.0	13.63
72	68.8	8.48	45.9	10.98
77	100.3	6.61	65.0	8.62
82	132.3	5.17	93.4	6.59
Person-years exposed to risk in the whole group	11,364.5		7,785.0	

otherwise abstainer," and "heavy occasional, otherwise moderate," (d) heavy steady. In Table XXIX, for females, only the first two of these categories are included, because of the infrequency of occurrence of any heavy drinking females in the material. In these tables the values are not carried beyond age 82, because of the paucity of data at the higher ages. In computing the expectations of life, however, all available data were used, and the exposure to risk figures at the bottom of each table contain all persons in the category, including those over 82.

Considering first the males, it is seen from Table XXVIII that it is the moderate steady group which has the lowest age specific death rates. At all ages except the last tabled (82)

the q_x values for this group are lower than those for any other drinking category, including the abstainers. The persons in the moderate occasional group show unexpectedly high age specific mortality rates. They are even higher, age for age, than those of the heavy occasional group up to, and including, age 57, though the differences are not large after age 52. The most reasonable interpretation of this unexpected and apparently incongruous result is to be gained, I think, from the fact that the persons in this group under discussion are *essentially abstainers,* with only an occasional very moderate indulgence in alcohol. Looking at them for the moment in this way, they appear to be the worst moiety of the abstainer class from the longevity standpoint. Their age specific death rates are higher than those of the strictly abstaining group up to, and including, age 57. The inclusion of this group with the moderate drinkers has the effect of making the q_x values for the " all moderate " class higher than they would be if that class included only steady or regular moderate drinkers. If they were put in the abstainer class, where they really belong, the male moderates would then show lower death rates than the abstainers at all ages, just as the females do.

The heavy occasional group resembles the abstainer group fairly closely in its mortality rates up to the age of 57. Thereafter its death rates are higher than those of the abstainers. This suggests that an occasional heavy indulgence is probably not serious in its effects upon longevity until middle life, when it tends to become so.

The heavy steady group shows a bad mortality rate practically throughout, as would be expected. This group includes the persons who, quite literally, drink themselves to death. From age 72 on their rates are lower than those of the heavy occasional class, but in both cases the experience at these ages is too small to give any dependable rates.

In the females, as shown in Table XXIX, the age specific death rates are lower in the moderate occasional group than in the moderate steady group up to, and including, age 57. Thereafter they are higher. Throughout the period covered the rates for the moderate steady group are lower than those for the total abstainer females (*cf.* Appendix Table 2). But from age 67 on the rates for the moderate occasionals are higher than those for abstainers.

Taking the material as a whole the highest expectations of life throughout are experienced in the moderate steady group, in the case of both males and females.

The general conclusion of the whole study up to this point is plainly that this material affords no evidence to support the view that moderate drinking in any way impairs the duration of life, however measured. On the contrary moderate drinkers, and particularly moderate steady drinkers, show a small but consistent superiority in longevity to any other class of persons within the present material. Heavy steady drinkers exhibit a definite impairment of life expectancy, as compared with all other classes, over the age range where the experience is sufficiently extensive to warrant confidence in the results.

With these definite and clear-cut results in hand, the task remaining is, first of all, to see what can be done in the direction of finding a reasonable interpretation for them, supported by definite evidence. The results are unquestionably novel. So far as concerns the effect of moderate drinking, they run counter to general preconceptions and prejudices. But these preconceptions and prejudices rest, in some measure at least, upon the not inconsiderable body of actuarial data in the literature of alcohol. Is this apparent contradiction real, or, on the other hand, are the present results congruent with general insurance experience when properly interpreted?

To such matters as these the remaining chapters of this book

will be devoted. Before leaving the discussion of the life tables, however, there are some other points regarding which the data need to be put into the record. Throughout this book reference has frequently been made to the fact that the present material differs actuarially from the general population in the way it does, when considered as a whole, chiefly because it is a collection of *family* statistics, in which parents necessarily occur in much greater proportion to the whole than they do in any general population. It has been shown earlier that the mean duration of life is higher in parents than in the general population, as would of course be expected because in the class "parents" there can have been no deaths of infants, children, or youths. But it is important to examine more particularly into the actuarial characteristics of a large random sample of parents in general, in order to see whether these characteristics do or do not deviate from those of a general population, in the same sense that the present material used in the study of the effect of alcohol has been found to. To this end Tables XXX and XXXI are presented at this point.

These are life tables calculated for fathers as a group (Table XXX) and mothers as a group (Table XXXI). The data on which the computations were based are derived from a collection of genealogical material in the archives of the Institute for Biological Research of the Johns Hopkins University. The records were taken from five standard, and critically compiled, genealogical works (200–205). In extracting the data from these volumes every individual and every family group was taken down to the time (roughly in the eighties of the last century) when persons still living began to appear in the records. In other words, no material was used from these genealogies involving families in which some of the persons in the family were dead and some living. All persons in all families were

TABLE XXX

LIFE TABLE FOR FATHERS (GENEALOGICAL RECORDS) AS COMPARED WITH GENERAL POPULATION (GLOVER)

Age x	Death rates — $1000q_x$		Survivors — l_x		Expectation of life — $\mathring{e}_x$		Difference in expectation ($\mathring{e}_x$) over general population. Years
	Fathers	White males O.R.S., 1901	Fathers	White males O.R.S., 1901	Fathers	White males O.R.S., 1901	
30	1.68	7.99	100,000	100,000	41.10	34.88	+6.22
35	3.31	9.32	98,806	95,824	36.56	31.29	+5.27
40	5.33	10.60	96,797	91,203	32.27	27.74	+4.53
45	8.02	12.63	93,786	86,169	28.23	24.21	+4.02
50	11.25	15.37	89,551	80,420	24.44	20.76	+3.68
55	15.00	21.18	84,049	73,704	20.88	17.42	+3.46
60	20.41	28.59	77,183	65,224	17.51	14.35	+3.16
65	28.83	41.66	68,636	55,105	14.38	11.51	+2.87
70	41.12	58.94	57,884	43,022	11.59	9.03	+2.56
75	60.00	88.43	45,502	30,030	9.07	6.84	+2.23
80	89.00	133.53	31,655	17,223	6.94	5.10	+1.84
85	132.00	191.76	18,179	7,374	5.23	3.81	+1.42
90	193.00	262.78	7,925	2,138	3.76	2.85	+ .91
95	265.00	349.71	2,288	369	2.79	2.12	+ .67

included without selection when all of them were dead, and the records were complete. The data include, therefore, only completed lives. In point of time they include persons of American colonial stock living in the seventeenth, eighteenth, and nineteenth centuries. In Tables XXX and XXXI the parents are weighted with their fertility. In the present connection this is believed to be the fairest procedure, because the present material is composed of persons exhibiting a fertility much above the average of the general population as a whole. Fertility and longevity are known to be correlated. But it should be said that life tables for parents unweighted with their fertility do not differ signifi-

TABLE XXXI

LIFE TABLE FOR MOTHERS (GENEALOGICAL RECORDS) AS COMPARED WITH GENERAL POPULATION (GLOVER)

Age x	Death rates — $1000q_x$		Survivors — l_x		Expectation of life — $\mathring{e}_x$		Difference in expectation ($\mathring{e}_x$) over general population. Years
	Fathers	White males O.R.S., 1901	Fathers	White males O.R.S., 1901	Fathers	White males O.R.S., 1901	
30	4.24	7.72	100,000	100,000	39.89	36.42	+3.47
35	7.53	8.39	97,168	96,053	35.98	32.82	+3.16
40	9.06	9.31	93,160	91,944	32.42	29.17	+3.25
45	9.34	10.63	88,921	87,535	28.84	25.51	+3.33
50	9.40	13.37	84,845	82,565	25.11	21.89	+3.22
55	11.60	18.69	80,707	76,480	21.27	18.43	+2.84
60	16.50	25.06	75,555	68,689	17.55	15.23	+2.32
65	26.20	36.41	68,299	59,288	14.15	12.23	+1.92
70	41.60	53.69	57,992	47,648	11.21	9.59	+1.62
75	64.30	80.39	44,943	34,325	8.75	7.33	+1.42
80	95.00	121.15	30,306	20,774	6.76	5.50	+1.26
85	139.00	174.60	16,876	9,676	5.15	4.10	+1.05
90	193.00	245.32	7,142	3,143	3.77	3.02	+ .75
95	265.00	335.71	2,075	606	2.78	2.21	+ .57

cantly from Tables XXX and XXXI, in that portion of life here treated. A complete discussion of these life tables for parents will be published elsewhere. Here the parts of the tables from age 30 on will alone be presented, because it is the portion which is directly comparable with the life tables of the present material regarding the alcohol problem. The functions tabled are the life table death rates (1000 q_x), the survivorship (l_x), and the expectation of life ($\mathring{e}_x$). For comparison the corresponding functions from Glover's Tables VII and X (whites in the Original Registration States, 1901) are exhibited. The total life (person) years exposed to risk in the case of fathers (Table XXX) is 585,012.5, and in the case of mothers (Table XXXI) 570,492.5.

These life tables show that parents, as a class, are markedly different actuarially from the general population. Not only is their average duration of life greater, as would be expected from the fact that as a class they are free of mortality tax until roughly an age of 15 to 20 years is reached, but Tables XXX and XXXI show that both fathers and mothers have lower specific death rates, at all ages from 30 on to the end of the life span, than do the corresponding moieties of the general population. This fact is a demonstration of the remarkable biological selection, relative to health and constitution, which the assumption of parenthood entails. While in a general and vague way it has been known that such a selection must occur, and that the environmental factors associated with married and parental life tend towards greater longevity than do those associated with bachelorhood and spinsterhood, the extent and magnitude of action of these factors have not before been measured, so far as I am aware. It suggests extremely interesting and important actuarial considerations, from the viewpoint of life insurance practice, which cannot be gone into here.

The magnitude of the deviations of the life tables for parents from those for general populations, is even more significant than appears on its face. For it is to be remembered that these genealogical data are mainly derived from a period of time when general population death rates were much higher than they were in 1901, the time at which a general population table is chosen for comparison here, for purely practical reasons. The fall in general American death rates since 1700 has been marked. This fact would, of course, tend to make the differences between parental and general populations shown in Tables XXX and XXXI, a *minimum*.

In order to make clearer the significance of these facts for the present discussion Figs. 16 and 17 are introduced. These

are plots of the survivorship lines, starting with 100,000 persons at age 30, for (a) the abstainer group in the present material, (b) the moderate drinker group in the present material,

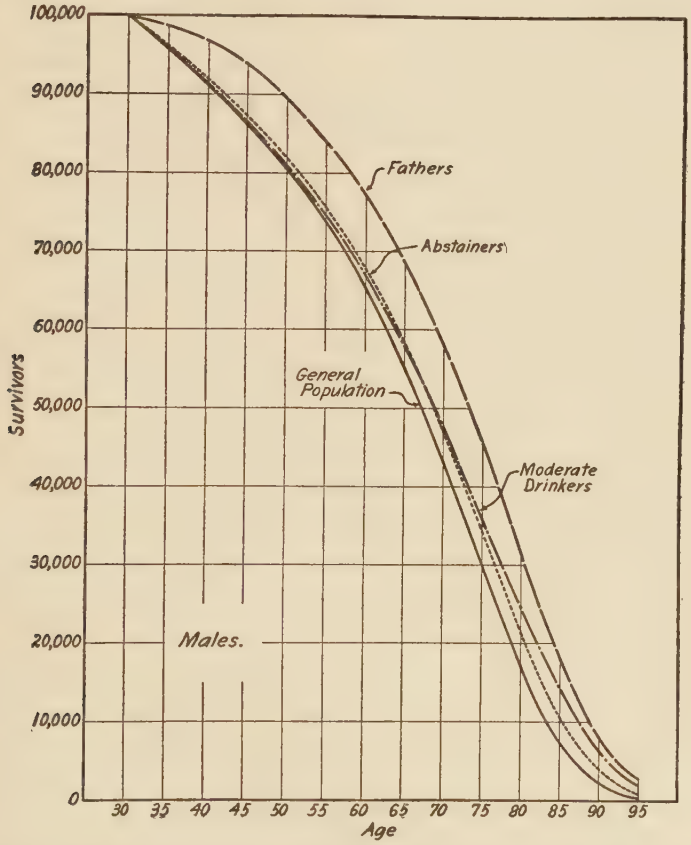

FIG. 16. Survivorship curves for males. Solid line — general population (males in cities, O.R.S. 1901); dash line — fathers; dot line — abstainer group in present material; dash-dot line — all moderate group in present material.

(c) whites in cities in the Original Registration States (data for (a), (b) and (c) taken from Table XXV), and (d) parents

(data taken from Tables XXX and XXXI). Fig. 16 deals with males, and Fig. 17 with females.

Figure 16 shows that the males in the present material, used

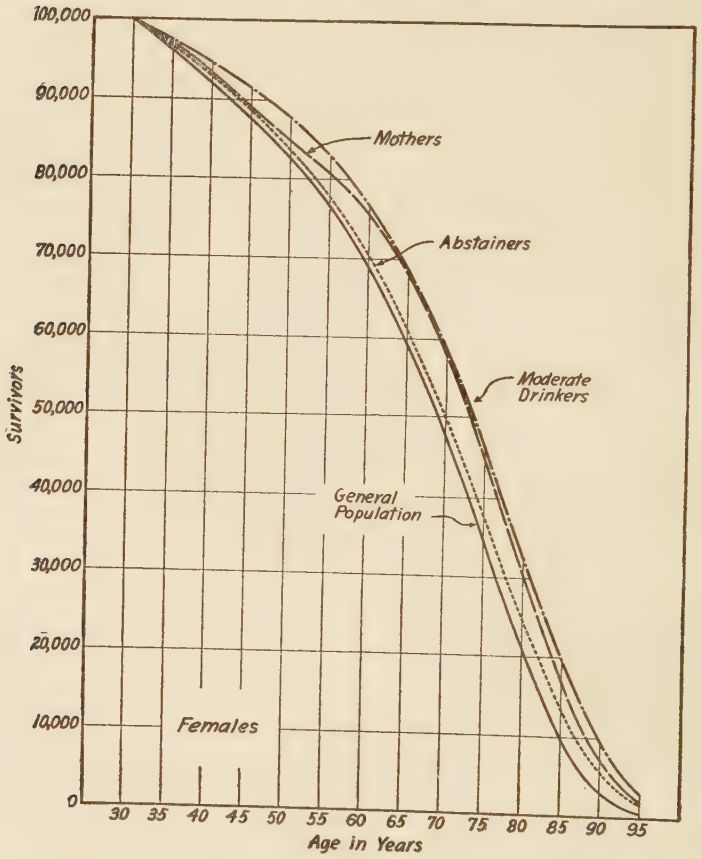

FIG. 17. Survivorship curves for females. Significance of lines as in Fig. 16.

for the study of the effect of alcohol, show a deviation from the general population, actuarially, in precisely the same direction as do a group of fathers as a class, and to an intermedi-

ate degree. As has been repeatedly pointed out in what has preceded, this result is exactly what would be expected from the fact that the present material is derived from family statistics, overloaded with parents, as compared with a general population.

In the case of the females, as shown in Fig. 17, essentially the same relationship holds, but there is a complication introduced by the fact that in the genealogical material from which the life tables for mothers are derived there was a much higher degree of fertility than in either the present alcohol material, or in the general population of cities in 1901. The consequence of this exaggerated fertility (by present standards) was an excessive mortality from child-birth, and its after effects, in the age periods from 30 to 60. The survivorship line for mothers is, as a result, closer to the general population line throughout, and especially up to age 60, than is the corresponding line for fathers in Fig. 16. The moderate drinker line lies above that for the mother group throughout, insignificantly so above age 60, but markedly between ages 30 and 60. It is possible that the moderate drinker group of females in this material is a less good sample, for some reason I have been unable entirely to fathom, than the corresponding group of males, and perhaps exaggerates the difference between moderate drinkers and abstainers in life expectancy. But even making all due allowance for this, and taking all the evidence into account, the conclusion seems firmly established, for the present relatively large and homogeneous experience, that moderate drinking does not sensibly or significantly increase mortality, as compared with complete abstention from alcoholic beverages.

It has been shown that parents, as a class, have a lower rate of mortality than the general population at all comparable ages. Furthermore it has been asserted that the present mate-

rial may be expected to include an excess of parents, because the basis of its collection is the family unit. It now remains to demonstrate that this latter statement is true in fact.

Unfortunately there are no statistics showing the number of parents at any given time in the living general population of the United States. The nearest that official statistics come to furnishing the desired information is in the census figures for persons married, widowed, and divorced, which, taken altogether, state the number of living persons who, at the time of the census enumeration, have ever been married. These figures are not directly comparable with the data for number of parents in the present material, for the simple reason that not all married persons are parents, or ever will be. There are sterile marriages. Reynolds and Macomber (269) have lately made a careful review of the statistical evidence regarding the matter. They estimate that about 10 per cent of all marriages are sterile. This may be regarded as a careful, informed, and conservative opinion.

In Table XXXI *bis* are given the following data: (a) the percentage of persons of known ages in the entire population of the United States at the Census of 1910 who were married at the time, plus those persons who were either widowed or divorced (data from *Abstr. 14th Census,* 1923, pp. 224–225). These percentages include colored as well as white persons. (b) The percentage of abstainers in the present material who were fathers or mothers. (c) The percentage of moderate drinkers in the present material who were fathers or mothers. (d) The percentage of heavy drinkers in the present material who were fathers or mothers. In all groups these percentages are given for five broad age classes. These start with 25 years. I should have preferred to start with age 30, since that is the age at which all our life tables begin. But the available Census age classes exerted a constraining influence, as usual.

TABLE XXXI *bis*

SHOWING THE PROPORTION OF PARENTS IN THE MAIN DRINKING CLASSES OF THE PRESENT MATERIAL, COMPARED WITH THE PROPORTION OF THE GENERAL POPULATION EVER MARRIED (MARRIED + WIDOWED + DIVORCED)

Age Group	Percent in general population ever married		Abstainers. Per cent of		All moderate drinkers. Per cent of		All heavy drinkers. Per cent of	
	Males	Females	Fathers	Mothers	Fathers	Mothers	Fathers	Mothers
25–34	64.6	79.0	59.6	82.4	69.2	79.6	58.5	87.5
35–44	83.1	88.5	87.1	91.9	82.4	90.2	75.6	85.2
45–54	88.7	91.4	89.4	95.0	92.3	98.1	86.7	87.5
55–64	91.5	92.9	94.5	99.4	98.3	98.8	90.1	80.0
65 and over	93.4	93.4	100.0	99.7	99.5	100.0	96.5	80.0

The figures of Table XXXI *bis* show that the contention made throughout this book, that the present material contains disproportionately more parents at all comparable ages than does the general population, is certainly true. The abstainer and all moderate groups contain proportionately more parents, at all but two ages, than the general population contains of persons ever married. This means that the excess in respect of parents in the present material over the general population must be considerably larger than these figures indicate, because of the fact that 10 per cent of all marriages are sterile, on Reynolds' and Macomber's estimate already referred to. The excess of the percentages for the three drinking classes over the general population is generally higher among the females than among the males.

It is interesting to note that the proportionate numbers of parents are lower in the all heavy group of the present material, than in either the abstainer or all moderate groups. This is in line with the general experimental finding of various work-

ers that large doses of alcohol tend to reduce fertility. But with all due allowance for the smaller proportion of parents among the heavy drinkers, as compared with the other parts of this present material, it is still probable that even in the heavy drinking class of the present statistics there are proportionally as many parents as in the general population, or possibly even more.

The fact that our heavy drinking group contains proportionally fewer parents than the abstainer and moderate groups, would tend to make this group exhibit higher death rates at ages than the other two. From this consideration it follows that the poor showing of the life table for the all heavy group in this material, cannot be wholly due to the deleterious biological effect of the heavy consumption of alcohol. Probably it is mainly due to this cause, but the composition of the group relative to parentage probably also plays some rôle in producing the observed results.

In the discussion of the genealogical material in the present connection, the point is not overlooked that the particular sample of that material here used, even though it is a composite of five different sets of records, may be actuarially super-standard as a whole. But other unpublished studies which have been made upon it do not indicate that such is the fact. It is impossible in the space available in this book to go into this point in detail.

CHAPTER VI

EARLIER EVIDENCE ON ALCOHOL AND MORTALITY

The results set forth in the last chapter are in some degree manifestly in contradiction to a widespread popular belief regarding the effect of alcohol upon longevity. While this material clearly shows that heavy drinking has a deleterious effect upon mortality and longevity, it indicates with equal precision, no such effect of moderate drinking. Instead the mortality among moderate drinkers is generally the same as among total abstainers, or less, and the average longevity is greater.

It becomes now necessary to consider the meaning of these results, and to attempt to find a rational interpretation of them. To these tasks this and the following chapter will be addressed. The first step will obviously be to review critically the literature on the problem, in order that an understanding may be gained of the origin of the popular impression which is apparently so far at variance with the results of the present study.

The literature discussing the relation of alcohol consumption to mortality may be fairly divided into three general categories as follows:

A. Propaganda against alcohol, which presents no original observations or data, and through ignorance or design selects only such old data as are favorable to its predetermined conclusions.

B. Original material gained from the experience of life insurance companies.

C. Other original data relevant to the problem.

As would be expected the bulk of the recorded literature falls in Class A, and need not detain us long. Its general conclusion is that alcohol, in any amount or dilution, and regardless of all other considerations, invariably shortens life. After laboriously working through all of the literature in the field, it is my judgment that the titles in the bibliography at the end of this book numbered from 79 to 112 inclusive, may justly be put in this A category. If anybody wishes to read these contributions the bibliographic citations are here provided. Many more titles of the same sort might be listed, but this seems a reasonable sample.

Original actuarial studies of the effect of alcohol upon mortality begin with Neison (113). I have fully reviewed his original paper in (30) and therefore need only point out here that he demonstrated, by methods quite sufficiently accurate for the purpose, that *heavy* drinking definitely and seriously increases mortality and shortens life. The present results agree perfectly with this finding. Heavy drinking shortens life now just as effectively as it did three-quarters of a century ago. Neison dealt definitely and specifically with heavy drinkers only, just as I have done in the present study in the "heavy drinkers" of the experience. Because of this circumstance the results of Neison have significant value, in settling that part of the problem which relates to heavy drinking. Another more recent study showing the deleterious effects of immoderate drinking is included in the monumental treatise produced by the German Imperial Statistical Office entitled "*Krankheits-und Sterblichkeitsverhältnisse in der Ortskrankenkasse für Leipzig und Umgebung. Untersuchung über den Einfluss von Geschlecht, Alter und Beruf.*" The portion of the study relating to the effects of heavy drinking has been abstracted in a detailed manner by Holitscher (141). The heavy

drinkers showed a much higher mortality than the general population of moderate drinkers with which they are compared.

When we consider the effects of moderate drinking the work done prior to the present study will not so well withstand criticism. The evidence is all indirect, inferential, and not probative. I shall review it in some detail, however, because it does represent, in a rough way, a variety of different sorts of human experience with alcohol, and because it is only by setting forth in a comprehensive manner the results of this experience that we can, in my judgment, see the correct relations to it of the results of the present study. In this review I shall follow the lines laid down in (30).

At the outstart I should like to quote directly what I said in opening the discussion referred to (30, p. 261): "In my opinion much of this indirect and inferential evidence is interesting, is potentially significant, and must be taken account of in any scientific discussion of the problem of alcohol and mortality. But, from the standpoint of cold-blooded scientific objectivity — the single-minded desire to get at the truth regardless of what the moral, social, economic or other consequences or implications of that truth may be, in supposition or in fact — the trouble with all this indirect evidence on alcohol and mortality is that from its inherent character it cannot possibly prove anything, no matter how much it may be multiplied in amount. It merely suggests or implies what may be so. It has been repeatedly put forward by interested reformers, as completely probative, indeed to an extent that it is now generally so accepted by the public mind. But, in plain fact, it is only suggestive, not probative, and quite certainly in some cases suggests a wrong rather than a right conclusion."

Opinions of Physicians

The general impression of physicians has long been that alcohol plays a considerable part in mortality. This opinion has been based upon the consideration both of deaths directly and solely due to acute or chronic alcoholism, and of deaths in which alcoholism is supposed indirectly to play a contributory part. Fernet (114–116) collected and reviewed the experience and opinion of Paris physicians on this point with the following results:

Renon and Gaillard, reporting from two general hospitals, found alcoholism to be the principle cause of death, through delirium tremens, acute renal disorders, etc., in 23, or 10.26 per cent, of 224 deaths. In their opinion alcohol was an accessory or contributory cause of death, in lowering resistance to acute infections, in from 15 to 20 per cent of cases in general hospitals. Joffroy, in 1904–06, showed that alcohol was either a principal or contributory cause at St. Anne's Asylum in 17, or 43.18 per cent, of 44 deaths. Seglas, at the Bicetre, found it a principal or contributory cause in 46.31 per cent of 95 deaths. Fernet concludes from this evidence that alcoholism is an even more important factor in deaths in insane asylums than in those occurring in general hospitals. Joffroy and Seglas contributed similar data for the deaths of 1906 and 1907. The former found alcoholism a cause, principal or accessory, in 11, or 57.89 per cent, of 19 deaths, and Seglas found it a principal or accessory cause in 39, or 44.83 per cent, of 87 deaths. The summary of data from nine general hospitals showed alcoholism as principal cause in 108, or 10.2 per cent, in 1059 deaths, and as the contributory cause in 217, or 23.61 per cent, of 919 deaths. Legrain, in charge of the special service for alcoholics at Ville-Everard, reported that 25 of 27 deaths, in a year on

this service, were directly or indirectly due to alcoholism. Pfister (117) made an intensive study, in 1908, of the deaths in Basel attributed to alcohol, directly or indirectly. He contends that the Basel statistics on alcoholism are particularly good, having been kept for a long time uniformly and conscientiously by the same official; they deal with a community that is easily surveyable and yet of fair size (the population in 1900 was about 100,000). The statistics include also data privately submitted to the government by the physicians, not only on the deaths directly attributed to alcohol but also on deaths in which alcohol was supposed to play an indirect or contributory part. Between 1892 and 1906 inclusive there were 7,287 deaths among males over 20 years of age, of which 760, or 10.5 per cent, were thought to be due, directly or indirectly, to alcoholism. During the same time there were 7,733 deaths among females over 20 of which 115, or 1.5 per cent, were thought to be caused, or contributed to, by alcoholism. Between 1902 and 1906 inclusive, alcoholism was thought to have played a rôle in 3.9 per cent of the deaths at age 20–30, 12.2 per cent at age 30–40, 19.7 per cent at age 40–50, 15.9 per cent at age 50–60, 11.3 per cent at age 60–70, 5.9 per cent, at age 70–80, and 11.9 per cent at all ages over 20 taken together. During the same period in females, alcoholism played a rôle in 0.6 per cent of the deaths at age 20–30, 2.1 per cent at age 30–40, 3.0 per cent at 40–50, 2.3 per cent at 50–60, 1.8 per cent at 60–70, 0.6 per cent at 70–80. Of the 760 male deaths attributed to alcoholism, 174, or about one-fifth, died directly of chronic alcoholic intoxication or delirium tremens, one-seventh died of diseases of the digestive tract including cirrhosis of the liver, and another seventh of diseases of the excretory organs, 38 committed suicide, and 52 died through accident, these cases forming 8.9 per cent of 583 deaths from accident occurring in males over 20 during the period. Fifty-eight died of pneu-

monia, 30 of nephritis, 29 of infectious diseases, and 92 of tuberculosis.

Others who have discussed the mortality directly or indirectly attributable to alcoholism include Tamburini (118) (who reported a progressive decline between 1905 and 1910 in Italy in the death rate from chronic alcoholism, cirrhosis of the liver, and accidents caused by inebriety, all taken together); Hatton (119), Hindhede (120), McNally (121), Stevenson (122), and Vernon (123).

There have been two general investigations of the opinions of physicians as to the significance of alcoholism in mortality. The first of these is the report of the Committee of the Harveian Society (124), and the second a memoir by E. B. Phelps (125, 126). The Harveian Society's report included 10,000 deaths, classified as follows:

A. Deaths in no wise due to alcohol	8598
B. Deaths accelerated or partly caused by its abuse	1005
C. Deaths wholly due to it	397

The report concludes that of the 10,000 deaths there were "as nearly as possible 14 per cent in the causation of which alcohol appears to have played some part."

Phelps' investigation was more carefully planned. He took a copy of the Ninth Annual Report of the Bureau of the Census of the United States, containing the report of the mortality during the year 1908 in the Registration Area, and got a medical director of one of the leading life insurance companies to check off each cause of death "therein listed in the cause of which, in his judgment, any male deaths between the ages of twenty and seventy-four inclusive, could possibly have been due in whole or in part to alcohol." There resulted 106 causes selected out of the whole list. Then a jury of physicians was

appointed, consisting of the late Dr. Brandreth Symonds, Chief Medical Director of the Mutual Life Insurance Company of New York; Dr. Eugene L. Fisk, then Medical Director of the Postal Life Insurance Company, and Dr. William L. Gahagan, Medical Director of the United States Casualty Company. Each of these physicians had had private and hospital practice prior to assuming the named positions, each of them had published on medical subjects, and each of them had given especial attention to the relations of alcohol and mortality. These three men were asked to state independently their personal estimates of the percentage of male deaths from each of these causes, directly or indirectly due to alcohol, between ages twenty and seventy-four inclusive.

The results of applying the average percentages obtained from these three medical directors to the total mortality are shown in Table XXXII. (On next page.)

Phelps finally reaches a figure of 11.2 per cent as against the Harveian figure of 14 per cent of a generation earlier, and concludes that there has probably been a real diminution, during that period, in the mortality attributable to alcohol.

Little weight can be attached to the results of these two investigations (and still less to the British Medical Association Committee's Inquiry in 1885–1886, reported by Isambard Owen (127)). The chief reasons for this view are:

1. What these inquiries really estimate is not the true point at issue, namely the effect of alcohol upon the incidence of mortality or morbidity, but rather in an indirect and rough way, the relative prevalence of different drinking habits in the community. This is clearly and overtly the case in the Harveian Society's inquiry, and in somewhat less evident form in Phelps' study.

2. Physicians' estimates of the percentage of deaths due to alcohol, from any particular cause, or from all causes, are

TABLE XXXII

A Recapulation of the Total Number, and Number between Ages Twenty and Seventy-four, Inclusive, of Male Deaths in the Registration Area of the United States in 1908 from each of the 106 Causes; the Number of Male Deaths between Ages Twenty and Seventy-four, Inclusive, from these Causes Directly or Indirectly Due to Alcohol in the Basis of the Average Percentage Estimates of the Medical Directors of Three Leading Insurance Companies; and the Ratios of these Deaths to (1) the Total Number of Male Deaths at all Ages from these Causes and (2) to the Number of Male Deaths between Ages Twenty and Seventy-four, Inclusive, from these Causes.

(From Phelps)

Causes of death in each group.	Three groups of deaths from causes in which alcohol may have been principal causative factor, or contributory factor.	Total Male Deaths from these causes		Male Deaths between ages 20–74 inclusive presumably due directly or indirectly to alcohol		
		At all ages	Between ages 20–74 inclusive	Number	Ratio to Male Deaths	
					At all ages	Between ages 20–74 inclusive
28	Causes of death in the case of which alcohol may have been an important contributory factor, and sometimes the principal causative factor	107,586	73,747	18,337	17.0	24.9
48	Causes of death in the case of which alcohol may have been a minor contributory cause, or at least a distinctly disturbing factor	174,244	107,422	13,664	7.8	12.7
30	Causes of death in the case of which alcohol was not primary or secondary cause, but may have been a harmful contributory factor . .	38,300	17,689	852	2.2	4.8
106	Causes of death . . .	320,130	198,858	32,853	10.3	16.5

simply a rough measure of medical ignorance of the effects of alcohol upon the living organism. Take the plainest case: One of the Phelps' jury estimated 80 per cent, and another 90 per cent, of all deaths from cirrhosis of the liver to be due to alcohol. But if anyone will take the trouble to study the existing literature with some care, he will have grave doubt as to the real significance of alcohol in the etiology of this disease. The fact is that, in the first place, cirrhosis of the liver has been produced experimentally with alcohol only in the rarest of instances. In the second place, extreme and chronic inebriates exhibit cirrhosis of the liver, at autopsy, in only a small percentage of cases. The third member of Phelps' jury estimated 30 per cent of deaths from this cause to be attributable to alcohol. Which one of the three was right? In the present state of ignorance regarding the etiology of cirrhosis of the liver, no one can say more than that probably all were wrong. The average of the three estimates was 66.7 per cent. Does this average represent a scientific truth — a measurement? To ask the question is to reveal the absurdity of the whole case.

Statistical Comparisons of Alcohol Consumption and Mortality

The idea must have occurred to many statisticians to compare the consumption of alcohol per head of population with the death rate of the same population, either on the basis of different countries or cities, or on the basis of different times. Since the war, and its associated restriction of the sale of alcoholic beverages in various countries, there have been a number of studies of this sort. Thus Hindhede (120) argues that the fall in the Danish death rate in recent years has been due to restrictions of the alcohol traffic in that country. But there are many other factors which presumably have played a part

in the decline, and there is no way to determine statistically what proportion, if any, is justly to be assigned to reduced alcohol consumption. The death rate has fallen in this same period in other countries in which nothing has been done to restrict alcohol consumption. And, conversely, MacNally (121) reports that deaths from alcoholism reviewed in the Coroner's Office of Cook County, Illinois, have increased since national prohibition went into effect.

The fundamental difficulties and fallacies involved in any attempt to get light on the problem by crude statistical comparisons of mortality trends are well shown in Fig. 18. This diagram compares (a) consumption of alcoholic beverages reduced to an absolute alcohol basis per head of population in gallons, from 1880 to 1908 inclusive, and (b) crude annual death rate from all causes, per hundred of population, in the Registration Area of the United States between the same dates, except that prior to 1900 the mortality figures are available only at decennial intervals.

The diagram is plotted on an arithlog scale in order that slopes may be directly comparable.

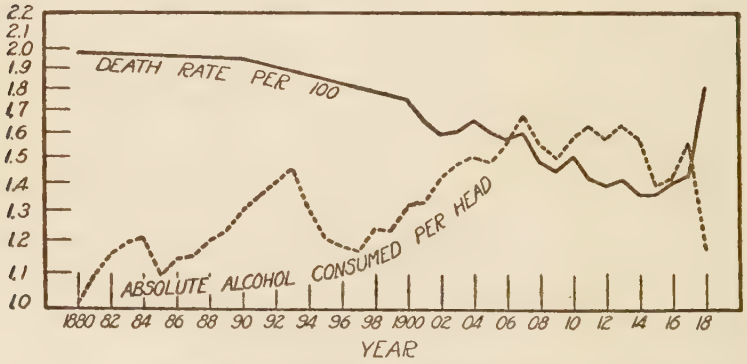

FIG. 18. Comparison of consumption of absolute alcohol per head of population, and crude death rate per 100 population in the United States Registration Area, 1880–1918 inclusive.

From 1880 until about 1906 the consumption of alcohol generally rose, and the death rate just as definitely declined. From 1906 on to the epidemic year, 1918, the two curves ran a course suggesting, but not entirely fulfilling, a condition of parallelism. In 1918 the alcohol consumption abruptly dropped, as a result of war-time prohibition, and the mortality rate shot up, because of the influenza pandemic. This last year plotted exposes the essential weakness of all such crude statistical comparisons. In that particular year it is definitely known why the two curves took the course they did, and furthermore, unless one is prepared to maintain that war-time prohibitory legislation was the cause of the influenza epidemic, or, on the other hand, that the epidemic was the cause of the prohibitory statutes (passed, incidentally, some time before the outbreak of the influenza), he will be forced to the conclusion that there is no causal connection between the movements of alcohol consumption and mortality in the year 1918.

An ingenious statistical study of the problem on an international basis was presented some years ago by the distinguished Danish-American statistician and actuary, Arne Fisher (128). He determined the correlation between consumption of alcohol per head and the male mortality rate. His data are given in Table XXXIII.

Applying to the data of Table XXXIII the usual method of measuring correlation, by the calculation of the Galton-Pearson coefficient, the values shown in Table XXXIV were obtained, together with their probable errors.

All the correlations are insignificant, having regard to their probable errors. In the earlier ages the coefficients are negative and of higher values than the remainder. If these figures could be taken at their face value they would indicate that mortality prior to age thirty-five or forty tends to be lower

TABLE XXXIII

CONSUMPTION IN LITERS PER HEAD OF 50 PER CENT SPIRITS AND BEER, AND THE MORTALITY RATES OF TWELVE COUNTRIES

50% Alcohol	Beer	Male Mortality per 100,000 of Population by Age							Country
Liters	Liters	25	30	35	40	45	50	55	
1.02	2.0	676	673	716	850	1056	1364	1877	Italy
2.31	7.8	710	710	760	910	1130	1460	1980	Finland
2.87	33.0	887	770	737	713	916	1096	1446	Norway
3.97	43.9	372	422	525	685	830	1101	1542	New Zealand
4.07	55.6	448	519	633	816	1083	1395	1816	Australia
4.17	121.6	454	566	732	931	1233	1657	2308	Great Britain
5.51	79.6	645	754	901	1053	1277	1585	2095	United States
6.89	47.8	628	604	637	757	925	1124	1526	Sweden
7.12	101.3	513	556	697	922	1244	1693	2357	Germany
7.16	29.4	492	475	537	679	898	1177	1686	Netherlands
8.82	40.0	752	786	842	1104	1363	1701	2153	France
10.44	84.0	404	447	528	689	938	1187	1707	Denmark

TABLE XXXIV

CORRELATION COEFFICIENTS CALCULATED FROM THE DATA OF TABLE 33

Ages	50 per cent Spirits. Coefficient of correlation	Beer. Coefficient of correlation
25	$-.30 \pm .26$	$-.55 \pm .20$
30	$-.27 \pm .27$	$-.33 \pm .26$
35	$-.11 \pm .29$	$+.04 \pm .29$
40	$+.01 \pm .29$	$+.18 \pm .28$
45	$+.10 \pm .29$	$+.17 \pm .28$
50	$+.07 \pm .29$	$+.12 \pm .28$
55	$+.03 \pm .29$	$+.03 \pm .29$

as the consumption of alcohol per head of the population increases.

Again, however, probably all such studies as this can have but little scientific value in the elucidation of the problem of

the influence of alcohol on mortality. Statistical comparisons between alcohol consumption per head and death rate, however skilfully treated mathematically, leave out of consideration so many important factors as to make their results of no more than suggestive value at best. Before such correlations as those of Fisher could approach probative significance, the figures would have to be corrected for racial and occupational influences on mortality, to mention only the most obvious defects. The same considerations apply to such comparisons as the one shown in Fig. 18. What all such elementary statistical approaches suggest is that if a critical study could be made, by the method of partial correlation, it would probably show that there is no significant correlation between the alcohol consumption of a population and its death rate, when other relevant variables are held constant. This is only another way of saying that all the existing evidence indicates that other factors are vastly more important than alcohol in determining the magnitude of the death rate in any large population aggregate, under present conditions of existence.

Mortality in Different Occupations

It has been shown by many investigators that the persons engaged in the liquor trade, including manufacturing, wholesale and retail distribution, exhibit a tendency towards higher rates of mortality than persons in various other occupations. (*cf.* Farr (129); Emminghaus (130); Gutstadt (131); Andrae (132, 133); Bl. H. (134); Medico-Actuarial (135); Hoppe (136); Tatham (137); Hunter (98) (100) (138) (139); Deuchar, Sprague, and Low (140); Jacquet (112); Bertillon (142); Vernon (143); Neison (144); Scott (145); etc.).

The fact of this higher mortality has been interpreted to mean; first, that persons engaged in these occupations are, in

general, excessive consumers of alcoholic drinks; and, second, that their higher mortality is a result of the consumption of alcohol in excess. The papers cited cover English, American, French, Scottish, and German experience. Anyone interested can find the detailed evidence in the papers cited. It will not be presented here because of lack of space, and because none of it seems to have any particularly significant bearing upon our problem, for the following reasons:

1. No account is taken of the various hazards inherent in these occupations, except that of drinking alcoholic beverages. Before anyone can accept these figures as demonstrating anything about the effect of alcohol *per se* upon mortality, he would want some evidence as to the mortality of *abstaining* inn-keepers, brewery employees, etc. No such evidence is forthcoming.

2. No account is taken of the occupational selection factor involved. Do persons engaged in the liquor trades come from stocks which constitutionally are poor insurance risks? There is some indirect evidence that they do. In so far as this is the case, alcohol has nothing directly to do with their higher mortality rate.

3. The material is widely heterogeneous in respect of drinking habits. These occupations do not represent a homogeneous group of excessive drinkers.

Life Insurance Experience

The first place to which one naturally turns in a desire to obtain accurate information regarding the effects of alcohol upon mortality is the experience of the great life insurance companies. The casual notion of most people certainly is that this experience must have long since settled the question, and that such an investigation as the one with which this book

deals is really a work of supererogation. Unfortunately — and when I think of the time and labor I have spent on the problem it is with deep feeling that I say unfortunately — this is not the case.

There are two fundamental reasons why the question is not to be solved by this simple and easy route. The first reason is that inquiry promptly discloses the fact that the total amount of first-hand data regarding the matter, derivable from insurance figures, is much smaller than would *a priori* have been supposed to be available. There is a vast amount of second-hand literature based upon the small quantity of first-hand actuarial contributions. A sufficient portion of this feeble stuff was referred to at the beginning of this chapter. But obviously it is only the basic, original papers which are of real significance.

The second reason is that the relatively small amount of original actuarial data that is in this literature, is sadly lacking in critical relevancy to the problem, and particularly in specificity of appositeness.

The first important original contribution based upon the regular experience of an insurance company was made by Neison (146) in 1889. His results and conclusions have been excellently summarised in the report of the Advisory Committee of the Control Board (Liquor Traffic) (147), pp. 122–123, and may be here quoted from that source:

"Comparing the Rechabites (a society of total abstainers) with the Odd Fellows and Foresters, a difference very similar to that found in the United Kingdom Temperance and General Provident Institution is seen. The Rechabites had only 69 per cent of the deaths calculated on the basis of the Foresters' experience, and their expectation of life at age 30, was more than 4 years longer than that of the Foresters. This comparison, however, probably overstates the advantage, as the Rechabites'

statistics cover the period 1878–1888, while the Foresters' data were for 1871–1875 and the Odd Fellows' 1866–1870. With regard to sickness the average weeks of sickness claim per member were higher among the Rechabites up to the age groups 40–50, but lower for the ages 50–60 and 60–70 than in the non-abstaining societies. The average number of weeks of sickness, not per member, but per member sick, were about the same among Foresters and Rechabites, and the actuary concluded that the higher general rates at earlier ages among the Rechabites were due to less perfect medical examination at entrance, his reason being that new entrants showed a much higher percentage of sickness claims than members of the same age, but longer standing in the Society. This, which is contrary to the usual experience of Friendly Societies, pointed, in his opinion, to less careful selection of entrants."

It was pointed out in the same place (p. 123) that these observations are subject to certain obvious criticisms. Again we may quote:

"(1) The Rechabites were a very much smaller society than either of the non-abstaining societies; both the Odd Fellows and the Foresters statistics covered in each case more than ten times as many years of life and more than fifteen times as many deaths, as the Rechabites, while the comparison does not relate to the same calendar years. (2) Both occupational and geographical distribution may be important factors of mortality, especially in industrial populations, and neither could be analyzed."

Moore (47) analyzed the experience, over a period of sixty years, of the United Kingdom Temperance and General Provident Institution. This company has operated in two sections, a Temperance and a General. The basis of the division is described by Moore as follows:

"Persons are eligible for the Temperance Section who do

not take alcohol as a beverage in any form. The continued adherence of the assured of the principles of abstinence is checked by an annual declaration to that effect. Such persons are described throughout this paper as 'Abstainers,' 'Temperance Lives,' or 'Teetotalers.' Persons who do not come within these conditions are eligible for the 'General Section' only, and are herein described as 'Non-abstainers.' If an Abstainer ceases to abstain he is transferred to the General Section, and, on the other hand, policy holders in the General Section who become Abstainers are generally eligible for transfers to the Temperance Section."

The essential result of Moore's comparison of this "temperance" experience (T^M) with that of drinkers (O^M) is shown for male lives in Table XXXV, which is condensed from Moore's Table XXII, p. 247. The data for drinkers were derived from the combined experience of 23 British insurance

TABLE XXXV

EXPECTATION OF LIFE OF MALE ABSTAINERS AND NON-ABSTAINERS (MOORE)

Ages	Expectation of life	
	T^M	O^M
10	55.021	51.459
15	50.973	47.323
20	46.949	43.183
25	42.967	39.083
30	38.827	35.067
35	34.595	31.159
40	30.328	27.360
45	26.100	23.668
50	22.016	20.107
55	18.130	16.722
60	14.554	13.571
65	11.338	10.716
70	8.491	8.208

companies. The values here tabulated are the expectations of life (mean after-life-time) at the indicated ages.

It will be seen that the abstainers enjoyed the greater expectation of life at all ages. The same is true, to an insignificant extent, of female lives, but the experience is much smaller. It is also true for those holding endowment policies.

The Medico-Mortality Investigation of 43 American and Canadian insurance companies (135) presented several sorts of data, of a somewhat fragmentary character. In general it was found, in all the comparisons made, that there was an excess mortality associated with alcohol consumption. But it should also be noted that the pertinent experience was not large, from an actuarial viewpoint. Furthermore it throws no light on the question of the effect of moderate, as distinct from heavy, drinking upon mortality, because the comparisons are between drinkers of all degrees as classified at entrance, on the one hand, and a general life-table including abstainers and drinkers of all degrees on the other hand.

Porter's paper (148) deals with the 1907–1912 experience of the Mutual Life Insurance Company of New York, classifying certain occupational groups of males according to drinking habits, as stated at the time of application for the policy. The results are shown in Table XXXVI.

The ratio figures of actual to expected mortality are not directly comparable between different investigations. The value of this ratio depends in part upon the number of "expected" deaths, which in turn is determined by the particular life table used in making the calculation.

Comparing the ratios of actual to expected deaths of Table XXXVI *inter se,* as is legitimate, it will be seen that the supposed abstainers exhibit a lower death ratio than the supposed non-abstainers at all ages, and in practically all groups, amounting in the total to 11.6 points.

TABLE XXXVI

MUTUAL LIFE OF NEW YORK EXPERIENCE, 1907–1912 (PORTER)

ON APPLICATION	Exposure	Deaths		Ratio
		Actual	Expected	
Class 224—Indoor Clerical Service				
Total class	39.657	90	192.110	46.9
Total abstainer	16.979	37	76.816	48.1
Temperate	19.926	44	99.790	44.0
Moderate	2.752	9	15.504	58.1
Class 226—Merchants and Dealers				
Total class	58.090	243	389.150	62.5
Total abstainer	16.565	42	104.400	40.2
Temperate	34.616	163	236.324	69.1
Moderate	6.909	38	48.426	78.7
Class 229—Salesmen (not in Liquor Business)				
Total class	32.157	97	159.918	60.6
Total abstainer	11.443	30	52.144	57.6
Temperate	17.985	55	93.256	58.9
Moderate	2.729	12	14.518	82.7
Class 233—Farmers				
Total class	65.399	243	374.109	64.9
Total abstainer	32.026	103	170.966	60.3
Temperate	32.315	133	196.122	67.8
Moderate	1.058	7	7.021	99.4
Class 240—Factories and Mills Proprietors, Managers and Superintendents				
Total class	15.718	76	114.365	66.4
Total abstainer	3.651	17	26.014	65.5
Temperate	10.133	53	74.645	71.0
Moderate	1.934	6	13.706	43.7
Total classes 224, 226, 229, 233, 240 combined				
Total class	211.021	749	1229.652	60.9
Total abstainer	80.664	229	430.340	53.4
Temperate	114.975	448	700.137	64.1
Moderate	15.382	72	99.175	72.7
Total non-abstainer	130.357	520	799.312	65.0

Phelps (150) presented, in 1915, a tabulation of the experience of the Northwestern Mutual Life, made by the actuaries of the company itself. The results are here reproduced as Table XXXVII. The mortality ratio is figured on the basis of monetary loss, involving therefore not only mortality *per se*, but the amounts of the several policies as well. It has, however, been repeatedly shown that the error involved in this procedure is negligible, if the whole experience is large.

TABLE XXXVII

MORTALITY EXPERIENCE OF THE NORTHWESTERN MUTUAL LIFE INSURANCE COMPANY ON ABSTAINERS AND NON-ABSTAINERS FOR THE YEARS 1886 TO 1895, INCLUSIVE, FIGURES TO THE END OF 1900 (PHELPS)

A = Abstainers; *B* = Beer or Wine Drinkers; *D* = Whisky Drinkers and a few large users of Beer or Wine.

	No. of policies	No. of deaths	Expected death loss	Actual death loss	Per cent of expected death loss
Ages 15 to 29					
A	47,293	1,298	$4,616,350	$2,321,656	50.29
B	17,156	512	2,063,651	1,204,635	58.37
D	3,977	140	630,643	378,449	60.01
Ages 30 to 39					
A	38,841	1,255	$5,755,681	$2,824,570	49.07
B	17,177	598	3,336,726	1,614,659	48.39
D	7,363	296	1,804,842	1,073,242	59.46
Ages 40 to 49					
A	15,802	830	$4,351,042	$2,212,873	50.86
B	6,838	405	2,546,517	1,391,599	54.65
D	3,952	285	1,845,384	1,157,160	62.71
Ages 50 to 59					
A	4,315	497	$2,518,685	$1,700,857	67.53
B	1,800	231	1,412,400	809,215	57.29
D	1,242	193	1,196,951	1,053,429	88.01

TABLE XXXVII — *Continued*

	No. of policies	No. of deaths	Expected death loss	Actual death loss	Per cent of expected death loss
Ages 60 to 69					
A	541	130	$667,418	$432,213	64.76
B	227	51	323,072	242,244	74.98
D	151	34	292,204	195,026	66.74
Ages 70 and upward (full paid under their new numbers)					
A	16	14	$12,953	$24,842	191.79
B	2	1	200	94	47.00
D	1	0	483	0	0.00
Total—All ages					
A	106,808	4,024	$17,922,129	$9,517,011	53.10
B	43,200	1,798	9,682,566	5,262,446	54.35
D	16,686	948	5,770,607	3,857,306	66.84

Mortality Experience with Non-Abstainers

Class B	Policies	Deaths	Expected loss	Actual loss	Per cent
Under age 40 . .	34,333	1,110	$5,400,377	$2,819,294	52.21
Age 40 and over .	8,867	688	4,282,189	2,443,152	57.05
Total	43,200	1,798	$9,682,566	$5,262,446	54.35
Class D					
Under age 40 . .	11,340	436	$2,435,585	$1,451,691	59.60
Age 40 and over .	5,346	512	3,335,022	2,405,615	72.13
Total	16,686	948	$5,770,607	$3,857,306	66.84

These figures indicate that the supposed wine and beer drinkers experienced substantially the same mortality as the supposed total abstainers, while both were well below the supposed whiskey drinkers. In so far as any insurance figures have scientific meaning as to the alcoholic habits of the insured throughout their lives, the beer and wine group of the Northwestern tabulation may perhaps be supposed to be moderate drinkers, in the usual sense of the words, and Class D heavy drinkers. Phelps is strongly of this opinion, but we are here on shaky ground.

Results similar to those quoted have been shown by the experience of the following insurance companies, among others: Sceptre Life, British Empire Mutual, Abstainers and General Office, Scottish Temperance Office, Scottish Imperial Office, Sun Life, etc.

Lounsberry (151) reports a small experience with a "total abstinence" class of risks in the Security Mutual Life Insurance Company of Binghamton, N. Y. An entrant in this class was required to have been a total abstainer for at least the last 5 years preceding entrance and was required to promise continued abstinence for the remainder of life. The experience covers 12 years, but the average duration of membership was only 3.42 years. The 6754 men in the total abstinence class had $37,470,602 exposed to risk. The expected loss was $378,-833. The actual loss was $185,660, or about 49 per cent of the expected, but it is impossible to judge what the 49 per cent means, in the absence of any information regarding the "loading" of the expectation.

Nicholl (152) has discussed the experience of the United Kingdom Temperance and General Provident since Moore's (47) paper. The record of the company as regards the percentage of actual to expected claims in the two sections is shown in Table 38, for each valuation period from 1866 to 1924.

TABLE XXXVIII

PERCENTAGE OF ACTUAL TO EXPECTED CLAIMS (UNITED KINGDOM EXPERIENCE, FROM NICHOLL)

Period	Temperance section	General section	Difference
1866–70	74.9	93.7	18.8
1871–75	70.7	105.1	34.4
1876–80	69.8	99.7	29.9
1881–85	70.8	91.6	20.8
1886–90	68.9	94.8	25.9
1891–95	71.3	99.7	28.4
1896–1900	73.8	90.5	16.7
1901–05	72.0	88.3	16.3
1906–10	65.7	83.3	17.6
1911–15	62.8	83.9	21.1
1916–20	74.4	82.6	5.2
1921–23[1]	53.5	72.2	18.7
1924	54.1	64.2	10.1

[1] Excluding war claims the percentages are 62.1 in the temperance section and 71.8 in the general section, with a difference of 9.7.

It will be noted the difference between the two sections has tended to grow smaller with the passage of time.

The fundamental difficulties with the insurance material for the determination of the influence of different degrees of alcohol consumption upon duration of life are these:

1. There is no definite knowledge of the alcoholic habits of the individual over any significant portion of his life. The only knowledge an insurance company has of any individual, speaking generally, is (a) the statements of the individual himself when he applies for a policy; (b) the continuance of his life as evidenced by payment of premiums, and (c) his death, as evidenced by a claim under the policy contract. Now granting that every applicant told the truth when he applied, the picture of his alcoholic habits then set down is, and can be, only of that time and the immediate past. But nothing is more

certain than that the drinking habits of some individuals change from what they were at the comparatively early age at which insurance was applied for, on the average. These habits may and do change in both directions. Some persons become heavier drinkers, others less heavy, than when they applied for insurance. So then, in fact, it may be taken to be the case that in the non-abstainer section of insurance experience such as we have cited, there is a mixture, in wholly unknown proportions of (a) persons who, for the major portions of their lives, have been total abstainers; (b) moderate drinkers; (c) excessive drinkers. There will also be the same three classes, again in quite unknown proportions, represented in the abstainer class in the experience of all companies except those, like the United Kingdom Temperance and General Provident, which requires an annual statement from the policy holder as to his continued abstention.

2. Since most insurance companies are believed, and in many cases known, to discriminate against persons using alcohol as a beverage in more than a certain (to the applicant unknown) amount or degree, an incentive is at once created for the applicant to understate the amount of his alcoholic indulgence. The discrimination may take the form of refusal to accept the risk, or an increased premium rate, or a reduced participation in the so-called bonuses or dividends. But in either case there is a powerful incentive for the applicant to make out as favorable a case as possible for himself.

These difficulties have been noted by many students of the problem. Emminghaus (153) went so far as to maintain that there were no critically accurate statistical data from which the comparative mortality of abstainers and non-abstainers could be determined. This, I think, is too extreme a statement. A fair appraisement of the situation would seem to be that what the most reliable of the insurance experience has estab-

lished beyond reasonable doubt is that abstainers, as a class, have a lower mortality than non-abstainers as a class. Farther than this the insurance data, in my opinion, will not permit us to go, in the way of safe and critical conclusions. What it has to offer upon the question of the comparative mortality of abstainers and moderate drinkers, can at the best be regarded as only suggestive of what may be the truth.

CHAPTER VII

THE INTERPRETATION OF THE EVIDENCE

The general results of previous work regarding the effect of alcohol upon mortality, as set forth in detail in the last chapter, sustain two general conclusions. The first is that heavy drinking certainly shortens life, on the average. The second is that if abstainers as a class be compared with non-abstainers, as a class, without further precise specification as to the amount of drinking by the non-abstaining persons, the mortality of the abstainers is generally lower than that of the drinkers.

How are the results of the present investigation, set forth in detail in Chapter V, to be interpreted in relation to this general body of earlier experience?

In the first place the present study agrees with all other experience so far as concerns the effect of *heavy* drinking upon duration of life. Indulgence of this sort and degree shortened life just as definitely in the present material as it has in the general experience of mankind on the point. So we can put this category of drinking to one side without further discussion. Everybody is agreed that heavy drinking is deleterious.

In the second place, it will be recalled that it was shown in the last chapter that one of the chief difficulties about the best of the insurance data (such as those from the United Kingdom and General Provident, the Medico-Actuarial investigation, etc.) was that these data made no sharp and clear-cut discrimination between different classes of drinkers. All that the insurance data are able to show is that abstainers, as a class, exhibit lower mortality rates than do non-abstainers, as a class. But a class of non-abstainers falls so far short of

homogeneity as to have but little significance relative to the problem in which the public interest really lies, namely the effect of *moderate* drinking upon longevity.

If, however, we compare abstainers, as a class, with non-abstainers, as a class, within the present material on which this book is based, we shall find no essential discrepancy between these figures and those of the insurance companies. Instead there is substantial agreement in principle. This is demonstrated in Table XXXIX, which again is condensed from one of the detailed life tables of Appendix II. In it are compared the life table death rates at ages ($1000\ q_x$) and the expectation of life ($\mathring{e}_x$) of males who were, on the one hand, abstainers, or, on the other hand, non-abstainers (*i.e.,* either moderate or heavy drinkers as defined in this book). This table has been computed simply by pooling together all male drinkers in the experience, regardless of the extent of their drinking, and comparing the resulting pooled figures with abstainers. The resulting information is precisely similar in character and significance to that derivable from the experience of the insurance companies reviewed in Chapter VI.

From Table XXXIX it is seen that, in the present material, the death rates of the non-abstainers (all drinkers), as a class, are higher than those of the abstainers, as a class, at all ages up to 70. From that point on, owing to the favorable death rates of the heavy drinkers at advanced ages in this material, the rates in the " all drinkers " group are lower than in the abstainer group. But it must be remembered that the exposure to risk is rather small in this experience at these advanced ages. Furthermore, it is in this region of the table that the family basis of the present statistics produces its heaviest effect. Altogether the indications of the table after age 70 cannot be regarded as having anything like the same degree of reliability as those for the ages before 70.

TABLE XXXIX

CONDENSED LIFE TABLES FOR (a) ABSTAINERS AND (b) ALL DRINKERS. MALES

Age x	Abstainers death rate ($1000\ q_x$)	All drinkers death rate ($1000\ q_x$)	Abstainers expectation (e°_x)	All drinkers expectation (e°_x)
30	7.54	9.19	36.34	33.60
35	8.33	11.66	32.69	30.22
40	9.63	14.44	29.06	27.05
45	11.62	17.61	25.47	24.07
50	14.61	21.31	21.99	21.24
55	19.07	25.78	18.64	18.55
60	25.79	31.42	15.49	15.99
65	36.03	38.89	12.58	13.55
70	51.85	49.25	9.97	11.25
75	76.65	64.34	7.71	9.10
80	116.10	87.35	5.90	7.14
85	157.92	123.73	4.55	5.45
90	214.80	175.28	3.44	4.06
95	292.17	248.31	2.54	2.94

The superior position of the abstainer group in its life table rates of mortality up to age 70, as compared with the non-abstainers, as a group, is not inconsiderable in degree. This is shown by Table XL, which expresses the differences in the

TABLE XL

PERCENTAGE EXCESS IN EXPECTED MORTALITY OF NON-ABSTAINERS OVER ABSTAINERS, ON THE BASIS OF DEATH RATES OF TABLE XXXIX

Age	Percentage which difference between Abstainer and All Drinkers expected deaths is of the Abstainer expected deaths. Equal exposure.
30	21.9
35	40.0
40	49.9
45	51.5
50	45.9
55	35.2
60	21.8
65	7.9

number of deaths between the two groups which would be expected if an equal number were exposed to risk at each age in each group, as percentages of the expected deaths among the abstainers.

It is thus seen, to take the maximal case as an example, that the number of deaths which would be expected in the non-abstainer group at age 50, if equal numbers at that age were exposed to risk in both abstainer and non-abstainer groups, is a little more than half again as high as the number of expected deaths in the abstainer group.

To summarize the evidence to this point, we may say that it has been demonstrated by Tables XXXIX and XL that *the present material leads to the same results as the general run of most trustworthy life insurance experience,* in respect of all the points on which the experience of insurance companies can furnish any evidence at all relevant to the problem of the effect of alcohol upon duration of life. These points are: (1) that heavy drinkers exhibit an extremely high mortality as compared with abstainers or with the general population, and (2) that non-abstainers, as a class, exhibit a higher mortality than abstainers, as a class.

So then it cannot be alleged against the material of the present study that it does not agree with general life insurance experience. It does, on every point in respect of which a real comparison can be made.

But, going back to the data presented in Chapter V, we *know,* for the present material, precisely why the death rates up to age 70 for the non-abstainers are higher than those for the abstainers, by the relatively large amounts shown in Table XL. It is simply because the non-abstainer group is not homogeneous in respect of alcohol consumption. It includes heavy drinkers as well as moderate drinkers. But, as has already been shown in Chapter V, moderate drinkers and abstainers

have substantially the same death rates (within the probable limits of fluctuation due to sampling) up to about 65 or 70. The bad showing of the non-abstainer death rates of Table XXXIX therefore is essentially due simply to the inclusion of the heavy drinkers in the heterogeneous non-abstainer group. But, again, as has already been pointed out, it is precisely this which is done when comparison of drinkers and non-drinkers is made on the basis of life insurance experience.

With the important point now settled that the present material is not in disagreement with insurance experience where there is parity of relevance, let us turn to further consideration of the moderate drinking group. It has been suggested by many persons, at different times, that the similarity in the mortality rates of abstainers and moderate drinkers, or the possible slight superiority of the moderates as compared with the abstainers, is mainly due to factors not connected directly with alcohol at all. This argument, in one form, runs as follows: The abstainer is really a poor risk, and knows it, or believes that he knows it. He therefore abstains from alcohol, thinking that it will do harm to his already poor health. On the contrary the moderate drinker is an average, healthy sort of person who is in fact a good insurance risk. It is then concluded that if the persons in the moderate group had been abstainers instead of moderate drinkers, they would have shown a greatly superior duration of life, on the average, to the real abstainers. The reason they have in fact only about the same, or a little higher expectation of life, is because their real vital superiority to the abstainer group has been curtailed by the harmful biological effects of their moderate drinking.

This same argument in somewhat different form has been discussed by Stevenson (122). He says (pp. 279–280):

" There remains for consideration Professor Pearl's finding, that moderate drinkers live slightly longer than abstainers.

In the first place, such slight differences as those shown by him at ages under 60 . . . need not be taken very seriously; and, in the second place, even if taken as significant, the result is perhaps not so very surprising. In discussion of this subject we cannot assume that, but for their habits regarding alcohol, the expectation of life of the abstainers and non-abstainers compared would, age for age, be equal. It may surely be assumed that total abstainers include amongst their ranks a larger proportion of the intemperate enthusiasts commonly referred to as 'cranks' than do non-abstainers. We have no data as to the natural longevity of cranks, but it may quite possibly be below the average. Some evidence pointing in this direction may be found in the record of the discussion of Moore's paper. Sir T. P. Whittaker, referring to a suggestion that abstainers were largely so because, being naturally vigorous persons, they did not feel the need for 'stimulant,' and so were better lives, used words which merit more attention in this connection than they have received.

"'Probably he knew more of the abstainers in the country than any man in the room, and he doubted if that were so. He doubted whether, apart from their abstinence, abstainers were a particularly careful, healthy and strong lot. They were a restless, strenuous, active, and sometimes a contentious lot, especially in years gone by. Men who took up an unpopular stand in social life were necessarily of that stamp of mind, and he did not think the strenuous worrying attitude of mind was necessarily conducive to health or longevity. It was the quiet, equable, steady man, who allowed nothing to worry him, that lived. The teetotaller was a strenuous, conscientious, active, fighting, worrying, driving sort of fellow.'

"Here we have a description of the temperament of the typical teetotaller by an undoubted authority in the form of a prominent champion of abstinence, which might stand for sym-

pathetic description of 'cranks' in general. Have such persons a normal expectation of life? We have no statistical evidence as to this, but I, for one, am disposed to accept Sir T. P. Whittaker's surmise on the point as very probably true. On the one hand, then, we have the combination of drink and the devil, and on the other hand of teetotallism and the crank, with the result that statistical measurement of the effect of alcohol upon mortality seems to be impossible.

"A further point to be considered is that men of the habits of Pearl's class of moderate drinkers may well be, on the average, exceptionally good lives. They are the men whose lives have proved their temperance and self-restraint in the use of alcohol; and it is probable that the same qualities also serve them in good stead in other connections. Some abstainers, on the other hand, are such, because either their family or their personal history has indicated the advisability, in their case, of total abstinence. There does not, then, seem anything surprising in the fact, if true, that very moderate drinkers, such as Pearl's, live slightly longer lives than total abstainers. This would even be compatible with some adverse effect of moderate consumption of alcohol upon longevity, for we cannot say how long the moderate drinking type would live under conditions of total abstinence."

Stevenson implies that it is not any easy matter to determine, from human material, the precise effect of alcohol apart from other factors. Every student of the problem will agree. The general argument for a selective factor in the case has caused me much thought. It presented a baffling puzzle to get some really critical and differentially relevant human evidence, which would carry us farther forward towards a sound judgment on the point. By considering, on the one hand, how one would go at the solution of the problem experimentally, and, on the other hand, turning back to my original family his-

tories, I hit upon a scheme which at any rate offers some new evidence that is critically relevant, though not as large in amount as might be wished.

If one were going to set up an experiment with guinea pigs, let us say, to determine the effect of moderate alcohol consumption upon longevity, as compared with total abstinence, the procedure would be essentially as follows: From each litter of the stock animals, pairs of the same sex, age, and general health would be chosen. These pairs of litter brothers (or sisters) would be like each in genetic constitution, if the stock were pure bred and substantially homozygous, or if the stock were large and had for a long time been bred wholly at random relative to all characters. Then one of each such pair of litter mates would be given a moderate amount of alcohol throughout life, and the other would be given no alcohol at all. In other respects the environmental factors, such as food, lodging, climate, etc., would be kept the same for each member of the pair.

It occurred to me to go back to the original records and see whether one could extract a sufficient number of pairs of brothers in which one of the pair had been a moderate drinker throughout life, and the other an abstainer, to enable the calculation of age specific death rates. If this could be done then we should have at least an approximation to the guinea-pig experiment described above.

It was possible to get 94 male abstainers who had 113 moderate drinking brothers. Each one of the 94 abstainers had at least one full brother in the moderate group. Some of them had more than one. Hence there is a larger number in the moderate group as a whole. From the standpoint of human vital statistics these are small numbers — much smaller than one would like to have. But the records regarding them are known to be accurate, and therefore they must be admitted

to have some value. It is also fair to point out that if the experiment described above were carried out with guinea-pigs, and 207 animals were used in the experiment, no biologist would question the results. On the contrary it would be acclaimed an unusually convincing research because such a large number of animals had been used!

In Table XLI the essential data, regarding these brother-brother pairs, are exhibited.

TABLE XLI

THE AGE SPECIFIC DEATH RATES (1000 q_x) FOR (a) A GROUP OF ABSTAINERS AND (b) A GROUP OF THEIR FULL BROTHERS WHO WERE MODERATE DRINKERS

Age	A. Abstainers who were full brothers of individuals in section B of this table			B. Moderate drinkers who were full brothers of individuals in section A of this table		
	Person-years exposure to risk	Deaths	Death rate (1000 q_x)	Person-years exposure to risk	Deaths	Death rate (1000 q_x)
20	401.5	2	5.0	536.5	3	5.6
25	304.0	2	6.6	420.0	6	14.2
30	220.5	2	9.1	302.5	3	9.9
35	168.0	1	6.0	237.5	2	8.4
40	134.0	4	29.5	189.5	4	20.9
45	91.5	2	21.7	139.5	2	14.2
50	65.0	1	15.3	88.5	2	22.3
55	45.0	..	....	56.0	..	....
60	23.5	2	82.0	33.0	..	....
65	8.5	..	....	7.0	..	....
70	5.0	..	....	2.5	..	....
75	.5	1	....	.....	..	....
Total	1467.0	17	....	2012.5	22	....

Owing to the fact that there happened to be no deaths among the moderate drinking brothers after age 50, although there were 98.5 person-years exposure to risk of dying, the death rates in the two groups can be compared only up to and including age 50.

This comparison may best be made graphically. This is done in Fig. 19, which plots the specific death rates for the two groups.

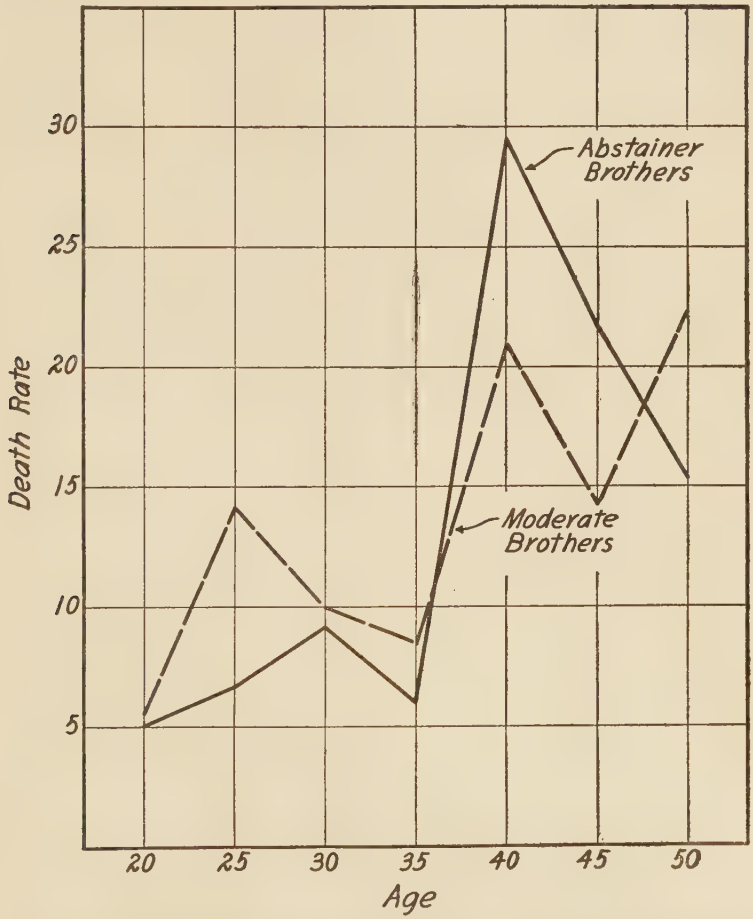

FIG. 19. The unsmoothed life table death rates for (a) a group of abstainer males (solid line); and (b) a group of moderate drinking males who were full brothers of the abstainers (dash line).

While the death rate lines of Fig. 19 are irregular, on account of the meagerness of the data, what evidence they do afford favors the view that the moderate drinking has not adversely affected duration of life. While the specific death rates up to age 35 are higher among the moderate drinking brothers than among the abstainer brothers, after that age they are generally lower. Having regard to the small extent of the experience the two lines run as closely similar courses as could reasonably be expected. There is furthermore every reason to suppose that these pairs of brothers are more nearly alike in genetic constitution than random pairs of males in general would be. While it is practically never possible in human biology to get data exactly corresponding to the experimental records of general biology in precision and control of variables, we nevertheless approach this ideal in the present instance fairly closely. And the result confirms the conclusion that moderate drinking does not sensibly shorten the duration of life.

In connection with the preparation of this book, I have compiled, with the assistance of Miss E. Marion Pilpel, an extensive annotated bibliography on the biological effects of alcohol, drawing widely on medical sources, as well as zoölogical, botanical and general biological. The general characteristics of this literature are of interest in the present connection. The first broad impression that one gets is that relatively little study, either by medical men or biologists, has ever been devoted especially to the effects of small or *moderate* alcohol consumption. The literature abounds in material on the results of excessive dosage. Its harmful effects have been demonstrated over and over again in all departments of the biological economy. But what of the clinical, physiological, and other biological studies on the effects of moderate drinking? Though relatively few in

number, it will be interesting and suggestive in the present connection, to examine a few of the more recent of them.

In 1903 Cabot (207) presented the results of a study of measurements of the blood-pressure before, during, and after the administration of alcohol to patients for the most part sick with typhoid fever, in the medical wards of the Massachusetts General Hospital. "We began our 4-hourly measurements," he says, "on 41 febrile patients recumbent in bed and upon unvarying diet. After 2 or 3 four-hourly records with each instrument (Riva-Rocci and Oliver) the patient was given either half an ounce of whiskey or an equivalent amount of alcohol properly diluted and flavored. This dose was repeated every four hours for the first 24 hours and then doubled and continued for 24 hours more [in most cases, though in a few it was continued several days]. After stopping the alcohol we continued the readings for at least 24 hours and often longer." Neither the maximum nor the mean blood-pressure showed any variations that could reasonably be referred to the action of the alcohol. So far as was determined by the methods and the cases used in this research, the action of alcohol upon the circulation was *nil*. The same neutrality and apparent inertness of therapeutic doses of alcohol in relation to the temperature, pulse-rate, respiration rate, appetite, sleep, delirium, and secretions (renal and cutaneous) of 309 patients suffering from a great variety of diseases, was the total impression derived from 2163 observations in these cases. Lieb (208, 209) got essentially the same results in 1915 in a similar study of patients at the Roosevelt Hospital. So also did Engelen (210) in 1921.

Dixon, who has done a great deal of careful, thorough work on the physiological effects of alcohol, showed (211) that: "In moderate doses and if well diluted alcohol has little effect

on the rate of the normal heart; in large doses it excites the medulla and slows the heart through the vagus. The failing heart is accelerated by alcohol. When taken by the mouth, in concentration, alcohol reflexly quickens the heart. In moderate doses alcohol causes dilatation of the superficial vessels and some slight constriction, which after large doses is followed by dilatation, of internal vessels. In moderate doses alcohol increases the activity and output of the heart. Reasons are given for suggesting that this effect is due, in whole or part, to the fact that alcohol is a readily assimilable food substance. In large doses, strengths over 0.5 per cent in the blood, alcohol depresses the heart; this is due to the direct toxic action of the drug. Alcohol in moderate doses administered to animals which show signs of circulatory failure, raises blood-pressure mainly on account of its effect on the heart. In normal animals and in man whilst the systolic pressure may rise a little or remain unchanged, the diastolic tends to diminish; in other words the difference between the systolic and diastolic pressure tends to increase."

Bickel (212) found experimentally that no dilatation of the heart followed the administration of moderate doses of alcohol, confirming the observation of Moritz on human beings in regard to this point. Some years later Bingel (213) found no evidence, in 48 German university students, of dilatation of the heart following beer drinking, even when coupled with strenuous fencing.

In relation to the incidence of diseases of the circulatory system, Mitchell, a county practitioner but apparently a keen clinician and observer, has recorded (214) some results of interest in the present connection. He analyses 118 deaths from cardio-renal disease, occurring during 20 years of his practice, and concludes that alcohol is an insignificant factor in the etiology of this group of diseases. Only 5 deaths in the lot

could be attributed in any way to alcohol. One hundred five out of the 118 persons were total abstainers, and 7 were moderate drinkers. Of 84 cardio-renal patients, alive and under treatment at the time of writing, 78 were total abstainers. Lian (215) found the incidence of arterial hypertension not significantly different in moderate drinkers from that in sober persons, though he presents evidence that heavy drinking is a factor of real importance in the causation of this condition.

Before leaving the discussion of the circulatory system I wish to refer to a paper by Kootataladse (216), which I have not been able to see in the original but only know from reviews in *Physiological Abstracts* and *Chemical Abstracts.* He carried out experiments on the isolated mammalian heart with dilute alcohol solutions and grape wine. The results with alcohol were not essentially different from those of other workers (*cf.* Starling (30), but the grape wine diluted to the same alcoholic content as the pure alcohol solutions, increased the rate 20 per cent, doubled the output and the strength of the beat, and increased the amount of fluid passing through the coronary vessels. A substance was isolated from the residue left after the distillation of the wine, which in a 1:500,000 concentration had a powerful effect upon the heart, similar to that of digitalis. The fresh grape juice does not contain this substance; it is formed only in the ripening of the wine and is a product of fermentation. Ordinary light wine contains about 0.0137 per cent of this active principle. These curious results need, of course, to be confirmed before they can be accepted, but they are in line with a fact long known and abundantly demonstrated, that the physiological effects of alcoholic beverages are not by any means always attributable to the ethyl alcohol content alone.

In this discussion it has not been my purpose to review completely what is known of the action of alcohol on the *cir-*

culatory system. The reader will find that well done in (29) and (30). All that I have attempted to do is to show by a few specific references that alcohol in moderate doses produces so little effect of any sort upon the circulation, in either the sick or the well, as to make it difficult to suppose that it could deleteriously affect life duration through this particular organ system.

Let us turn now to other aspects of the matter. Wrzosek (217) studied experimentally the influence of alcohol upon the growth of transplanted mouse carcinomata. The alcohol was given to the mice either by injection or with the milk used as food. The following was a typical experiment. Eleven young mice were given from the time of weaning 0.05 c.c. to 0.10 c.c. of alcohol daily with their milk. They were inoculated in the inguinal region with 25 mg. of tumor-broth. In 9 the tumor took. The average duration of life after inoculation was 36 days, and the average tumor weight at death was 7.4 grams. In 14 out of 15 control mice similarly inoculated the tumor grew. The average duration of life of the controls after inoculation was 28.5 days, and the average weight of the tumors at death was 7.3 grams. Wrzosek's general conclusion is that injection of moderate doses of alcohol had no deleterious effect upon these cancer mice. There was a slight diminution in the number of successful " takes " of the tumor following inoculation.

Tsurumi (218) has also studied the effect of alcohol upon mouse carcinoma, perhaps even more critically and intelligently than Wrzosek. Ten control and 11 alcohol-treated mice were inoculated with a rapidly growing strain of mouse carcinoma. The treated mice were then given daily 0.05 to 0.07 c.c. of 10 per cent alcohol in physiological saline by hypodermic injection. After something over two weeks of this treatment they were all killed with the following results:

Control

Total weight of 10 mice	152 grams
Mean weight per mouse	15.2 "
Total weight of tumors	11.8 "
Mean weight of tumor growth	1.18 "

Alcohol Treated

Total weight of 11 mice	165 grams
Mean weight per mouse	15.0 "
Total weight of tumors	7.59 "
Mean weight of tumor growth	.69 "

There was thus a definite inhibition of tumor growth in the alcohol mice. In fact most of the tumors in these treated animals grew very little. In another series of experiments Tsurumi studied the influence of alcohol on the formation of metastases in the lungs, following the intravenous injection of a rapidly growing mouse carcinoma. In 22 alcoholized animals only 22.7 per cent showed metastases on careful microscopic as well as macroscopic examination. But in 14 controls 64.3 per cent had metastases.

Meyer (224) made a thorough experimental study of the effect of moderate doses of alcoholic beverages on the secretory and motor activity of the *stomach.* No essential influence on the acidity of the stomach content could be made out with the doses used (15–30 grams of 45 per cent cognac or alcohol of corresponding strength). The alcohol was found to hinder somewhat the emptying of the stomach when the food was exclusively starchy, to have no influence when the food was purely protein, and to accelerate the emptying with fat.

Kast (225) used the Pawlow technique in a study of the effect of alcohol on the functional activities of the stomach in

dogs. He found that alcohol in moderate quantities, and in dilutions under 10 per cent, stimulates the stomach to an increased formation and secretion of hydrochloric acid. No increased production of mucus by the stomach could be observed at these dilutions. The most recent extensive and critical study of the effect of moderate consumption of alcohol upon the functional activity of the stomach is that of Haneborg (226). He shows that when the alcohol is taken with the meals there is an increased secretion of gastric juice, of higher hydrochloric acid content and greater digestive capacity.

Clopott (223) carried out a careful series of metabolism experiments with a moderate alcohol intake. His results entirely confirmed Atwater's as to the food value of alcohol. Of particular present interest in his conclusion that the alcohol had no demonstrable effect on the absorption of nourishment in the *intestine*.

Regarding the effect of moderate drinking upon the *kidneys*, and the excretory functions in general, there is little evidence available. Hultgen (220) studied the effect of heavy drinking (chronic alcoholism) on the kidney function, with fairly extensive and carefully recorded clinical data. Among 461 drinkers only 42, or 9.1 per cent, showed evidence of nephritis, and only 24, or 5.24 per cent, exhibited temporary or permanent albuminuria. McNider's (221, 222) recent experimental studies indicate that the prevailing impression of the excessive damage done to the kidneys by alcohol is exaggerated. In concluding a study of the effect of alcohol upon nitrogenous metabolism Mendel and Hilditch (23) say that perhaps the most significant impression which their data afford is the absence of pronounced alternations indicative of disturbed protein metabolism, even when the alcohol is continued for long periods of time.

In 1915 Reich (227) published a detailed study of the effect of different grades of alcohol consumption upon the defense mechanism of the blood against *infection*. He found that heavy consumption materially weakened these defenses, though curiously enough the phagocytosis of tubercle bacilli by human leucocytes was found to bear no orderly relation to the alcohol consumption of the individual. In all of the reactions studied very good and very bad reactions were obtained in some particular *individuals* in *all* the different groups relative to the amount of alcohol consumed. From this fact Reich concludes that alcohol is not the predominating influence in determining the effectiveness of the defense mechanism. In fact, so slight and irregular were the results, as between abstainers and moderate drinkers, that he found it impossible to say whether moderate drinking is capable of affecting these mechanisms at all. In this connection it is interesting to recall that Billings (228), in his analysis of the data got from questionnaires sent to a group of "brain-workers," found that 10.3 per cent of the total abstainers reported themselves as affected with "indigestion," or acute rheumatism, or nervous disease of some kind. Of the occasional drinkers 6.8 per cent reported themselves as thus affected, and of the regular, moderate drinkers 9.2 per cent.

The *growth* of moderately alcoholized plants and animals has been shown by a number of investigators to be better than that of non-alcoholized controls. Mast and Ibara (229) found that tadpoles in weak solutions of alcohol not only lived longer than the controls, but grew much larger in the same length of time; so much so that the superiority was apparent at a glance, and required no elaborate measurements for its demonstration. The differences are shown in the following extract from their data.

TABLE XLII

GROWTH OF TADPOLES (FROM MAST AND IBARA)

Date	Culture solutio	Average total length in mm.	Average length of body in mm.
May 6	2/3 per cent alcohol with plants	21.00	7.60
May 6	1/3 per cent alcohol with plants	18.55	6.86
May 6	1/3 per cent alcohol without plants	19.30	7.00
May 6	Tap water with plants	17.00	6.40
May 23	1/3 per cent alcohol with plants	19.24	7.14
May 23	Tap water with plants	16.31	6.44

Hecker (230) in his study of alcoholism in German school children found that while alcohol adversely affected growth in height, by the time 13.5 to 14.5 years of age were reached only 17 per cent of the abstaining boys were above the average height for the age (from Lange's tables) as against 45 per cent of the drinking boys.

Space is lacking for a further review of the literature on moderate drinking, but the general trend of it all may be summed up in this way. In the physiological, general biological, and clinical studies which have been made on alcohol, when the dosage is of the order of that involved in moderate drinking by human beings, there are extremely few results recorded which can be regarded as biologically deleterious. There are many results, well established by critical experimentation and observation, that indicate that small amounts of alcohol are beneficial in some biological processes. In others there simply is no detectable effect of small amounts of alcohol. All this is in the most extreme contrast to the large body of knowledge regarding the biological effects of large doses of alcohol. They are almost uniformly deleterious.

Many worthy persons who somehow seem unable to keep separate their moral convictions and their scientific activities have taken an extreme position, wholly unwarranted by the facts, to the effect that alcohol, in any quantity however minute, is always biologically harmful. An amusing commentary upon this position is afforded by the fact that ethyl alcohol is regularly and normally present in the blood and tissues of the living body. This has been demonstrated by Maignon (231), Ford (232), Reach (233), Schweisheimer (234), and others. The amount of alcohol so present is relatively minute. But Kühn (235) has recently shown, using an extremely exact interferometric technique, that the increase in blood alcohol noted after the administration of a single dose of alcohol, in the form of two bottles of beer taken with a large meal, is *smaller* than that occasionally observed after the administration of carbohydrate *without* alcohol. In his experiments the former averaged 0.0033 per cent (the maximum being 0.0051 per cent, or 17 interferometer drum units), while the latter attained a maximum of 0.0057 per cent, or 19 drum units.

Altogether I think that Starling's (30, p. 168) summing up of the case is entirely sound. It " depends upon the distinction between the use and abuse of alcohol. In moderation it is difficult to appreciate any harmful effects from its use, whereas when the temperance is abandoned and alcohol is used immoderately, its effects are evil and fraught with disaster to the individual and damage to the community."

CHAPTER VIII

THE RACIAL EFFECT OF ALCOHOL

Up to this point we have been solely concerned with the influence of alcohol upon the health and longevity of the individual. But there is clearly another and not less important purely biological aspect of the alcohol problem. What is the effect of alcohol upon the health and longevity of the race? Does its use bring about racial degeneration, ultimately to end in collapse and extinction? Or, is its use biologically beneficial to the race? To the consideration of these questions this chapter will be devoted.

During the past fifteen years more extensive and penetrating investigations have been carried out regarding the effect upon the descendants in the kinship nexus, of the use of alcohol by the ascendants, than in the entire prior history of the subject. These investigations have been in the main experimental, though a few extremely important ones have been statistical. It is the thorough application of the experimental method to the problem, however, which has markedly, and it appears securely, advanced our knowledge in this field. To get really critical, accurate and extensive statistics about any phase of the alcohol question from human beings is an extremely difficult task. It can only be done by enlisting the services of highly trained field workers, who, by visiting families, are able finally to establish such relations of friendliness, or even intimacy, as make possible frank and true statements about such a delicate question (in America, at least) as alcoholic habits. It is by this method that the results so far set forth in this book have been obtained.

In a laboratory experiment, on the other hand, not only are there no social or ethical elements to embarrass the research, but there is the opportunity of virtually complete control of the whole life of the individual. It is possible in this way to get a kind of evidence on the problem which can be gained by no other process.

The literature is extensive regarding the effect of alcohol upon the race. But fortunately it is easily and accurately divisible into two distinct portions. The earlier part, extending in point of time up to about 1910, need not detain us now, because it has been supplanted by the more critical work which has appeared since that date. The present era of the critical investigation of the problem of the racial effect of alcohol may be properly said to begin with the work of Pearson (236, 237, 238). The chief results of the first of these studies, which is the one directly and pertinently bearing upon our present problem, were as follows:

"There is a higher death rate among the offspring of alcoholic than among the offspring of sober parents. This appears to be more marked in the case of the mother than in the case of the father, and since it is sensibly higher in the case of the mother who has drinking bouts than of the mother who habitually drinks, it would appear to be due very considerably to accidents and gross carelessness and possibly in a minor degree to a toxic effect on the offspring.

"Owing to the greater fertility of alcoholic parents, the nett family of the sober is hardly larger than the nett family of the alcoholic.

"The mean weight and height of the children of alcoholic parents are slightly greater than those of sober parents, but as the age of the former children is slightly greater, the correlations when corrected for age are slightly positive, *i.e.*, there is slightly greater height and weight in the children of the sober.

In the case of the father the correlations are not significant having regard to their probable error; in the case of the mother they may be just significant but they are so slight as to have no importance.

" The general health of the children of alcoholic parents appears on the whole slightly better than the health of the children of sober parents. There are fewer delicate children and in a most marked way cases of tuberculosis and epilepsy are less frequent than among the children of sober parents. The source of this relation may be sought in two directions: the physically strongest in the community have probably the greatest capacity and taste for alcohol. Further, the higher death rate of the children of alcoholic parents probably leaves the fitter to survive.

" Parental alcoholism is not the source of mental defect in offspring.

" The relationship, if any, between parental alcoholism and filial intelligence is so slight, that even its sign cannot be determined from the present material.

" The normal visioned and normal refractioned offspring appear to be in rather a preponderance in the families of the drinking parents, the parents who have 'bouts' give intermediate results, but there is no substantial relationship between goodness of sight and parental alcoholism.

" The frequency of diseases of the eye and eyelids, which might well be attributed to parental neglect, was found to have little, if any relation to parental alcoholism.

" To sum up then, no marked relation has been found between the intelligence, physique or disease of the offspring and parental alcoholism in any of the categories investigated. On the whole the balance turns as often in favour of the alcoholics as of the non-alcoholic parentage."

It will be noted that there is here made for the first time the

definite suggestion that alcoholism in the parents may have a selective effect upon the offspring. The evidence for this conclusion is statistical in character, and consists of two points: (a) greater death rate among children of alcoholics, and (b) superiority of surviving children. There was no suggestion made of selection of germ cells in the gonads of drinking parents.

The most extensive experimental study of the problem is that which has been carried on for a number of years by Stockard (40, 239, 240). Earlier experimental work, such for example as that of Laitinen, and a number of others, had been felt to be open to rather obvious criticism, in one respect or another. Some researchers had used obviously faulty methods; others had not kept, or at any rate published, detailed records of just what was done or happened; others had failed to follow the matter far enough to justify any conclusions. All told, experimental work preceding Stockard's attack on the problem produced little effect on the mind of the scientific public.

Stockard's work with alcohol began in 1909, and was not at first genetic in character, but embryological. He instituted experiments on the modification of the development of fish embryos, which have their embryonic life outside the body of the mother. He has recently ((239) p. 255) reinterpreted the results of these experiments. In the original accounts of this work he did not lay the stress that he now does on the *selective* character of the results taken as a whole.

"When eggs were placed in water containing from 1 to 5 per cent of ethyl alcohol their rate of development was considerably inhibited and in the stronger solutions almost stopped. On later returning these eggs to the usual seawater environment development was resumed at a normal or almost

normal rate. Nevertheless, the arrests which had been experienced caused in many of the eggs the entire suppression of development, while in others the development of many organs and parts were greatly modified. The embryos developed abnormally and were monstrous in form.

"An important fact to be noted is the great individual variation in the way the eggs react to the solutions. When any lot of one hundred eggs are examined after these treatments it will be found that a certain percentage of them are developing perfectly normally, as if these had entirely resisted the effects of the treatment; others are only slightly affected; others exhibit more marked modifications; and a number of eggs have actually been killed. It is thus seen that the effect of the treatment varies with the hardiness of the egg. The poor eggs which would have, under usual conditions, developed into weak embryos are eliminated by the treatment, those of low average resistance are injured, while the particularly fine eggs are apparently not affected and live to hatch as vigorous young fish. This variable response occurs when eggs are treated with a sublethal dose of any chemical substance, and it is in no sense a peculiar reaction to alcohol.

"Another fact that should be fully recognized in drawing general conclusions from the responses of these embryos to alcohol is that exactly the same injuries and developmental deformities are readily induced by the use of a great number of other chemical stuffs, and also by unusual physical conditions. The fact that such deformities are experimentally produced in embryos by the application of alcohol does not in any sense demonstrate that human deformities have been induced by a similar cause. The amount of alcohol in the solutions to which the eggs are subjected is greater than could be tolerated in the human blood stream. It is also a fact that common salt (sodium chloride), sugar, and many other constantly used

substances in human food are equally as effective in inducing the same types of developmental modifications in these eggs as in the application of alcohol. The fact that an experimenter may use an effective dose of a certain substance to induce developmental disturbances has no direct bearing on the question of the use of this substance in very much smaller amounts by man or other animals.

" These results should be considered in an impartial manner and should not be distorted for one purpose or another in a discussion of the use of alcohol by man. I can only say, as an embryologist, that the experiments give no certain proof that alcohol has ever caused abnormal development in the human embryo. Neither do they prove that it may not, in so far as the doses here necessary to induce an effect are greater than we have reason to believe ever exist in the human body."

Following this work on externally developing embryos Stockard began the series of experiments on guinea pigs which has received such wide and deserved recognition. The initial purpose was to see whether in internally developing embryos results similar to those on fish eggs could be got. Alcohol was administered to guinea pigs by the method of vapor inhalation. This was a great advance in the experimental technique of the subject, because it avoided the secondary disturbances of general metabolism which may follow the daily administration of alcohol in solution *per os,* continued over long periods of time. It is the general experience of all students of the problem that animals receiving alcohol by inhalation remain in excellent general health, and in fact usually attain to a greater total duration of life than control animals not given alcohol. (*Cf.* Chapter I of this book.)

The general broad result of Stockard's studies, and essentially the only one in any way emphasized, or even recognizably

pointed out, in his papers over roughly the first ten years of the work, was that the administration of alcohol to the parents caused the production of a small, but still perfectly definite, proportion of defective offspring, many of which were non-viable for more than a short time, and some of which lived as monstrous forms. This aspect of Stockard's results has recently been illustrated by him in a table (239, p. 255) which may be reproduced here as Table XLIII.

TABLE XLIII

Alternate Matings of Normal Guinea Pigs, with Normal and Alcoholic Mates. (From Stockard.)

	Matings of 35 Normal Males successively with		Matings of 44 Normal Females successively with	
	Normal Females	Treated Females	Normal Males	Treated Males
No. of matings	81	81	77	81
Total offspring	196 2.42 Av. lit.	185 2.28 Av. lit.	195 2.53 Av. lit.	182 2.25 Av. lit.
Failure to conceive	6 7.90%	6 7.90%	3 3.89%	10 12.34%
Lived over 3 mos.	151 77.03%	105 56.64%	161 82.56%	118 61.83%
Died under 3 mos.	45 22.96%	80 43.35%	34 17.45%	64 35.16%
Defective	0	11 5.95%	0	9 49.7%

Table XLIII needs no particular comment. It demonstrates the main conclusion of all of Stockard's reports on this work prior to 1922. He has supported and developed it by a great variety of ingenious and painstaking experiments.

In 1914 Pearl (34 to 37 inclusive) began experimental work on the problem with the domestic fowl as material. The object was two-fold. Primarily there was the interest in seeing whether, by giving various volatile chemical substances over long periods of time by inhalation, it would be possible to induce *specific* and permanent changes in the genetic constitution of the germ cells — to induce and experimentally control new heritable variations, in short. In the second place Stockard agreed in conference that it was highly desirable to repeat his experiments with a different animal than the guinea pig, to see if the same results would be obtained. The methods used were in all essentials the same as those of Stockard. A somewhat detailed account of what these methods were has already been given in Chapter I, and need not be repeated here. This work started with a number of different substances, but presently came to the use only of ethyl alcohol. The investigation went on without interruption, and on a rather extensive scale, till the spring of 1917. A large amount of unpublished material had accumulated. My entrance upon war service in June, 1917, necessarily ended the experiments, but only for the time being, it was then expected. When in March, 1919, I was able to resume academic life, two developments of the work were contemplated. One was to publish as soon as possible all the mass of material which had accumulated in the experiments with alcoholized fowls and their progeny. The other was to continue the experiments with mice, and with all the refinements of method which had suggested themselves in the course of the earlier work. To this end a colony of mice of great genetic purity and homogeneity had been bred up by my assistant, Miss Sylvia L. Parker, during 1918 and 1919. Both of these projects received a summary quietus on November 24th, 1919, when my laboratory was completely destroyed by fire. Neither records nor animals could be saved.

The first year's work with fowls is summarized in Table XLIV. (From Pearl (36) p. 280.)

TABLE XLIV

SHOWING IN SUMMARY FORM THE EFFECT OF CONTINUED ADMINISTRATION OF ALCOHOL UPON THE F_1 PROGENY

Character Studied	Treated males × Untreated females	Treated males × Treated females	All Treated Parents	Untreated Controls	Net Result on Alcohol Offspring
1. Mean germ dosage index	137.8	299.0	210.35	0	
2. Percentage of infertile eggs (i.e., in which no embryos were formed)	25.2	59.2	41.7	25.3	−
3a. Percentage of embryos dying in shell	36.6	26.9	33.3	42.2	+
3b. Percentage of fertile eggs which hatched	63.0	12.3	66.7	57.8	+
4. Percentage of all eggs which hatched	47.1	29.4	38.6	44.4	−
5. Percentage mortality under 180 days of age	21.1	10.6	17.6	36.9	+
6. Percentage mortality over 180 days of age	5.9	13.6	10.3	15.3	+
7. Sex ratio : per cent males	48.9	45.5	47.7	50.0	0
8. Mean hatching weight per bird, males	34.91	36.97		34.24	+
9. Mean hatching weight per bird, females	35.04	37.17		34.73	+
10. Mean adult weight per bird, males	2669	2815		2392	+
11. Mean adult weight per bird, females	2020	2063		1928	+
12. Percentage of weak and deformed chicks	0.7	0	0.4	1.0	+
13. Abnormalities of Mendelian inheritance	0	0	0	0	0

This first year's work showed that out of 12 different characters for which there were quantitative data, the offspring of

treated parents taken as a group were superior to the offspring of untreated parents in eight characters.

These results were first presented in a paper read before the American Philosophical Society on April 15th, 1916 (241). It was suggested that these results, as well as those of earlier workers, might be most satisfactorily accounted for on the hypothesis that alcohol, and similar substances, act as selective agents upon the germ cells of treated animals. The essential points in the hypothesis were put in the following way.

(a) Assume that the relative vigor, or resisting power of germ cells varies, or grades continuously from a low degree to a high degree, and further assume that the absolute vigor of the whole population of germ cells is different for different species.

(b) In the intensity of dosage employed in inhalation experiments, alcohol does not destroy or functionally inactivate all germ cells but does so to some of them. The proportionate number of the whole population of germ cells which will be inactivated by such dosage may fairly be supposed to depend upon the mean absolute vigor or resisting power characteristic of the particular species or strain used. In a species with germ cells of absolutely low mean vigor proportionately more will be functionally inactivated than in a species of high absolute mean vigor of germ cells.

(c) Besides the germ cells which are wholly inactivated by the agent, and which we may designate as class (a), we may fairly assume that there is a possibility of two other classes existing, *viz.*, (b) germ cells which, while not completely inactivated, are so injured by the agent as to produce young which are measurably defective in some degree, and (c) germ cells which are not measurably affected by the agent at all in the dosage employed, and produce young which are not discernibly otherwise than perfectly normal.

(d) It appears entirely fair to assume that germ cells of the (a) class are of relatively the lowest mean vigor or resisting power, class (b) next, and class (c) the highest. The proportionate number of the two sorts of young, corresponding to classes (b) and (c) of germ cells, which would be expected to appear in any experiments made to test the point would clearly be a function of the mutual relationship or proportionality between two variables, the dosage of the deleterious agent on the one hand, and the mean absolute resisting power of the germ cells characteristic of the strain or species of animal used in the experiments on the other hand.

(e) If the dosage of the agent be relatively high in proportion to the mean absolute resisting power it would be expected that all the germ cells would fall into classes (a) and (b), producing no young at all, or offspring in some degree defective.

(f) If, on the other hand, the dosage, though absolutely the same, be relatively lower in proportion to the mean absolute resisting power of the germ cells it would be expected that all three germ cell classes, (a), (b), and (c), would be represented. The embryos actually formed would be chiefly produced by (c) germ cells, and to a much smaller extent by (b) cells. Under these circumstances it would necessarily follow that a random sample of the young produced after the action of the deleterious agent would, on the average, be superior in respect to such qualities as growth, etc., which may be supposed to depend in part at least upon germinal vigor, to a random sample of young formed before the action of the agent, because the germ cells of class (c) are a selected superior portion of the total germ cell population.

(g) Essentially that proportionality between effective dosage of the deleterious agent and absolute resisting power of the germ cells outlined in the preceding paragraph (f) is be-

lieved to have obtained in my experiments with fowls, Nice's experiments with mice (*cf. infra*), and nature's experiment with the workingmen's population, studied statistically by Elderton and Pearson.

In December, 1916, some of the results on the second generation of progeny were reported in the summary form of Table XLV.

Table XLV brings out the following points:

(a) Considering the total, and comparing chicks with any alcoholic ancestry, as a group, with chicks having no alcoholic ancestry, as a group, it is clear that there were fewer of the former than of the latter from a given number of eggs. In every 100 eggs from birds in the alcoholic series approximately 53 embryos were formed as against 80 in every 100 eggs in the non-alcoholic series.

(b) The total number of offspring dealt with in the experiments (1628) is respectable, and such as to lead to reasonable confidence in the results, especially in view of the fact that these results were in every essential particular in full accord as to their sense with those obtained in the 1915 experiments, and even more pronounced in degree.

(c) The prenatal mortality was approximately 10 per cent, and the postnatal mortality (to 180 days of age) was approximately 5 per cent lower in the chicks with alcoholic ancestry than in those of non-alcoholic ancestry.

(d) While from a given number of eggs 26.81 per cent fewer embryos were formed in the alcoholic series than in the non-alcoholic, 10.7 per cent more of these young were alive when adult age (180 days) was reached if their ancestry was alcoholic than if it were non-alcoholic.

(e) The great reduction in embryo formation (partial sterility) observed in the alcohol treated series appeared to be preponderantly due to the effect of alcohol upon the germ cells of

TABLE XLV

SHOWING THE MORTALITY (PRENATAL AND POSTNATAL) OF CHICKS OF ALCOHOLIC ANCESTRY, AS COMPARED WITH THOSE OF NON-ALCOHOLIC ANCESTRY

Matings in 1916

Nature of Ancestry	Number of Matings	Total Eggs Set	Total Embryos formed	Embryos formed	Pre-natal Mortality	Post-natal (180 day) Mortality	Chicks alive at 180 days of age
				per cent	*per cent*		*per cent*
Both parents alcoholic	5	82	18	21.95	11.11	25.00	66.67
Dam only alcoholic	3	63	5	7.94	80.00	0	20.00
Sire only alcoholic	13	512	274	53.52	47.08	13.79	45.62
Sire and one or more grandparents alcoholic	3	233	158	67.81	46.84	28.38	39.87
One or more grandparents alcoholic	3	187	113	60.43	46.02	6.56	50.44
All of alcoholic ancestry	27	1077	568	52.74	45.95	16.50	45.42
All of non-alcoholic ancestry	28	1333	1060	79.52	55.94	21.20	34.72
Differences	1	256	492	26.78	9.99	4.70	10.70
Alcoholic better = + / Alcoholic poorer = −		−	−	−	+	+	+

the female. The significant thing is the fact that the percentage of embryo formation steadily rises in the following groups: (1) Dam alone or dam and sire alcoholic, (2) sire alone, or sire and some other ancestry not including dam, alcoholic, (3) no ancestry alcoholic.

These results clearly indicated that the germinal selective action of the alcohol postulated in my earlier papers was continuing to work, so far at least as the basic biological viability (duration of life, mortality) was concerned. There was a mass of other evidence to this effect in the records which did not get published before they were burned.

It was shown, however, that there was no sensibly higher proportion of abnormalities of development among the embryos of alcoholic ancestry than among those of non-alcoholic ancestry. This is demonstrated in Table XLVI.

The results so far had been such as to indicate that alcohol did act as a selective agent upon the germ cells, and stress had therefore been laid upon this aspect of its action in interpreting the results. But it was fully recognized that alcohol might act similarly as a selective agent upon the developing embryos. In the summer of 1916, in fact, I carried out the first experiment of which there is record, so far as I know, specifically and solely directed to the testing of the question as to whether alcohol did act as a selective agent upon developing embryos (37, pp. 681–682).

Two incubators were used, each containing at the outstart 390 eggs. These eggs were selected with the utmost care to ensure likeness of age, of strain, and of other characteristics in the two lots. In one incubator 40 c.c. of 95 per cent ethyl alcohol were evaporated beneath the eggs daily. In the other incubator no alcohol was used. At the end of 7 days 130 eggs, designated as Lot 1, were removed from the alcohol incubator and allowed to complete their development in a normal non-

TABLE XLVI

SHOWING THE NUMBER AND PROPORTION OF CONGENITAL ABNORMALITIES AND MALFORMATIONS FOUND AMONG THE CHICKS OF ALCOHOLIC AND NON-ALCOHOLIC ANCESTRY IN THE SEASON OF 1916

Offspring of	No. of Matings	Mean Germinal Dosage Index		Dead in Shell after the Tenth Day of Incubation		Abnormal Killed at Hatch	Hatched Chicks		Total Chicks Surviving to the Tenth Day of Incubation and beyond	Total Abnormal	Per Cent Abnormal
		P.	G.P.	Normal	Abnormal		Normal	Abnormal			
Treated parents or grandparents or both.....	27	562.9	40.6	78	19	4	267	20	397	43	10.8
Untreated control parents .	28	0	0	190	23	14	372	42	641	65	10.2

alcoholic incubator. At the end of fourteen days the remaining eggs (after testing out infertiles) of Lot 2, which originally contained 130 eggs, were removed from the alcohol incubator and finished their development without further dosage of alcohol in a normal incubator. Finally Lot 3, originally containing 130 eggs, was subjected throughout the twenty-one days of incubation to the daily dosage of alcohol fumes. At hatching, all the normal chicks from both incubators were put together in the same house and brooder, and given throughout life the same treatment as to food, etc. Careful account was kept of mortality, each chick being marked to indicate the lot from which it came. The conditions of brooding were purposely made bad so as to obtain a maximum severity of post-embryonic environmental conditions, with a consequent high absolute mortality rate.

The result was that the more alcohol the embryos received during incubation the higher was the prenatal mortality, but until the dosage became so prolonged as to injure all the developing embryos, as in Lot 3, *the prenatal mortality was selective,* since the higher the prenatal death rate the lower was the postnatal mortality among the hatched chicks.

Immediately after the publication of these results, Danforth (242), in the spring of 1917, began experiments, which were not finally published until 1919, to see whether the selective action of alcohol upon the germ cells which Pearl had shown in respect of characteristics dependent upon general vigor, also obtained in respect of morphologic characteristics known to follow the Mendelian scheme of inheritance. He describes his experiments as follows:

" In each experiment a cross was made between normal, purebred stock on the one hand and hybrid, heterozygous birds on the other. The hybrid individuals were subjected for certain periods to two daily treatments with alcohol vapor, dur-

ing which time as far as possible all eggs laid were incubated. Since the original purpose of the work was to test the possibility of selecting germ cells rather than of modifying them through influences brought to bear on the soma, each experiment is brief and intensive.

"A control was obtained in each case by saving the eggs from the same flock, kept under as nearly identical conditions as possible, during a period before or after the alcohol experiment. All eggs that did not hatch were opened and the character of the contents recorded. Since the eggs were candled frequently many embryos were obtained only a few days after death and consequently in a good state of preservation. Very few of the dead embryos were found to be too macerated to afford the desired data. Some living embryos were purposely taken. For the sake of brevity, data obtained from eggs laid during the periods of alcohol treatment will be referred to as A, those from eggs laid in the control period as C. Each experiment, therefore, has an A and a C subdivision. In experiments 1, 2, and 3 the heterozygous individuals were males, in 4 they were females.

"The characteristics tested for their possible response to alcohol vapor were brachydactyly, polydactyly, and color."

The alcohol was administered by the inhalation method, as by Stockard and Pearl.

The results, which are analyzed and presented in detail with the most scrupulous care, can be given here only in briefest form. Table XLVII, which is Danforth's Table IV, summarises the data.

From these data Danforth concluded that there was definitely a selective action in respect of brachydactyly, which is the technical term for an abnormal shortness of the fingers or toes. "Polydactyly" means the occurrence of more than the usual number of fingers or toes. By treating a heterozygous

TABLE XLVII

PERCENTAGE DISTRIBUTION OF CERTAIN MENDELIAN CHARACTERS IN EMBRYOS AND CHICKS OF ALCOHOLIC (A) AND NON-ALCOHOLIC (C) PARENTAGE (From Danforth.)

EXPERIMENT		BRACHYDACTYL		POLYDACTYL		WHITE	
Number	Part	Observed	Expected	Observed	Expected	Observed	Expected
1	A	46.7 ± 3.1	39 ± 3.0	29.4 ± 2.7	36 ± 2.9	100	100
	C	39.2 ± 2.5	39 ± 2.5	33.2 ± 2.1	36 ± 2.2	100	100
2	A	43.3 ± 3.1	39 ± 3.0	37.9 ± 2.9	36 ± 2.9	50.0 ± 3.2	50 ± 3.2
	C	35.1 ± 3.7	39 ± 3.8	39.0 ± 3.7	36 ± 3.7	52.1 ± 4.0	50 ± 3.9
3	A	53.6 ± 3.4	39 ± 3.3	52.0 ± 3.4	36 ± 3.3	59.9 ± 3.5	50 ± 3.6
	C	42.7 ± 3.5	39 ± 3.5	40.1 ± 3.5	36 ± 3.4	56.5 ± 3.6	50 ± 3.7
4	A	56.3 ± 5.9	39 ± 5.7			42.8 ± 6.3	50 ± 6.4
	C	38.4 ± 2.9	39 ± 2.9			53.4 ± 3.1	50 ± 3.0
Totals	A	48.2 ± 1.8	39 ± 1.7	36.0 ± 1.7	36 ± 1.7	52.7 ± 2.2	50 ± 2.2
	C	39.0 ± 1.5	39 ± 1.5	36.0 ± 1.6	36 ± 1.6	53.9 ± 2.0	50 ± 2.1

parent with alcohol vapor of sufficient strength the proportion of brachydactyl to normal offspring was definitely increased in Danforth's experiment. For the other two characters studied the evidence is less probative, but indicates some selective action.

We are entitled to conclude from the large body of critical experimental evidence provided by Danforth and Pearl that, *in the fowl* certainly, alcohol can and does act as a selective agent, primarily upon the germ cells, and also can be made so to act in some degree upon the developing embryos. But since in the fowl the embryos normally undergo all but the earliest stages of their development outside the body of the mother, it follows that any selective action on the *offspring,* of alcohol administered to the parents in this form, must normally be mainly, if not entirely, germ cell selection.

We return now to Stockard. In 1918 (40) he published with Papanicolaou a long paper bringing his results up to date and discussing Pearl's work which had in the meantime appeared. In this paper he accepts Pearl's results in all essential particulars, and for the first time in his writing upon the subject definitely states that in the guinea pig alcohol acts in part as a selective destroyer of unfit and weak germ cells. He says (*loc. cit.* pp. 221–223):

" It seems to us in keeping with what is known of biological reactions in general and the guinea pig histories in particular to take the following position. The alcohol treatment acts on the germ cell population of both fowls and guinea pigs in such a manner that the weakest or least resistant ova and spermatozoa die from the effects of the treatment as germ cells without taking part in zygote formation. The somewhat more resistant ova and spermatozoa are greatly injured though still capable of forming zygotes. The zygotes, however, are so defective as to be capable of only a short period of development and die

during stages too early to be definitely detected by gross examinations of either the fowl's egg or the mammalian mother. Still other embryos are capable of development to later stages and are actually found dead, not only as the youngest embryos to be identified, but from these early stages there occurs a continuous series of prenatal deaths up to the full-term or still-births. Immediately after birth the post-natal mortality is greatest and gradually decreases until those specimens capable of reaching maturity often enjoy a comparatively long life.

" At the present stage of the two experiments it would seem as though this elimination of defective germ cells and very early embryos was much more intense in the fowls than in the guinea pigs as a group; so that the late prenatal and postnatal mortality among the fowl progeny was low and those specimens that hatched were the hardy survivors from this early vigorous process of germ cell and individual selection. The records from the double alcoholic and male treated lines among the guinea pigs forms a second step. The size of the litters and failures to conceive in these lines indicate a rather high degree of infertility or germ cell debility as well as early prenatal deaths, though this is not so extreme as among fowls, and the late prenatal and postnatal mortality is higher.

" Finally the female treated guinea pig lines produce large litters and have few infertile matings, indicating a low germ cell and early prenatal mortality, and here the late prenatal and postnatal mortality is highest, not entirely on account of the action of the treatment on the developing individual in utero since the same condition is found among other female generations than the one directly treated.

" This presentation of the situation is somewhat similar to that which Pearl has illustrated in his diagrams. The chief difference being that we would decrease the proportion of eliminated germ cells and increase the proportion of defective and

non-viable zygotes, and thus emphasize the selection of individuals rather than of germ cells."

With this interpretation I am in entire agreement. It should be noted that none of the evidence so far presented gives any indication, let alone proof, that alcohol produced any permanent, specific alteration of the germ plasm in respect of determiners or genes, as would be demanded by any supposition that an acquired character has been inherited. Stockard (240) is in agreement with this view.

In 1922, and subsequently, Stockard (239, 240) presented an analysis of his accumulated data strongly emphasizing the essentially selective action of alcohol in improving the racial characteristics of his guinea pig stock over the long period of the experiment. Table XLVIII gives the latest summary of his results.

Regarding these data Stockard (239, p. 257) says:

"Section I contains data from the control or normal lines. These animals are of the same blood lines as the alcoholic stock — brothers and sisters and parents and offspring are in the two groups, and actually in cases the same individuals have first been used in the control and later in the experimental groups. There can be no doubt that any original defect or weakness that may have been in the stock from which the alcoholic animals were derived must also have been in the control stock, since the stocks are in all cases actually the same. All records in the present tables are of pedigree animals from thoroughly known blood lines and relationships. There is no inbreeding in either the control or alcoholic lines. It is as certain as experimental evidence can be that any difference between the records of the control and the treated groups must be attributed to the action of the alcoholic treatment, since this is the only element of difference existing in the life histories or experiences of the two groups of guinea pigs.

TABLE XLVIII

THE RECORDS OF GUINEA PIGS OCCURRING IN DIFFERENT GENERATIONS OF ALCOHOLIC STOCK (From Stockard)

	Total No. and Litter Sizes	Lived over 3 months		Total Mortality under 3 months		Absorbed Premature Stillborn	Died within 3 months after birth
		No.	%	No.	%		
I							
Normal stock control:							
1	19	18	94.73	1	5.26	1	0
2	114	94	82.45	20	17.54	15	5
3	216	172	79.62	44	20.37	20	24
4	132	88	66.66	44	33.33	26	18
5	25	19	76.00	6	24.00	0	6
Average litter 2.72							
Av. litter weight 188.85g							
Totals	506	391		115		62	53
Percentages (mean) ..			77.27		22.72	53.91	46.68
II							
Alcoholic animals of all generations:							
1	58	45	77.62	13	22.37	9	4
2	316	241	76.26	75	23.73	51	24
3	579	364	62.86	215	37.13	137	78
4	204	78	38.23	126	61.76	83	43
5	40	11	27.50	29	72.50	22	7
Average litter 2.56							
Av. litter weight 170.66g							
Totals	1197	739		458		302	156
Percentages (mean) ..			61.73		38.26	66.22	33.77
III							
Alcoholic animals with treated parents (F_1):							
1	17	15	88.23	2	11.75	1	1
2	126	90	71.42	36	28.57	26	10
3	240	136	56.66	104	43.33	74	30
4	52	14	26.92	38	73.07	23	15
5	15	2	13.33	13	86.66	11	12
Average litter 2.56							
Av. litter weight 165.89g							
Totals	450	257		193		135	58
Percentages (mean) ..			57.11		42.88	70.31	29.68

TABLE XLVIII (*Continued*)

	Total No. and Litter Sizes	Lived over 3 months		Total Mortality under 3 months		Absorbed Premature Stillborn	Died within month after birth
		No.	%	No.	%		
IV							
Alcoholic animals with treated grandparents, great-grandparents, etc. (F 2-3-4):							
1	41	30	73.17	11	26.82	8	3
2	190	151	79.47	39	20.52	25	14
3	339	228	67.25	111	32.74	63	48
4	152	64	42.10	88	57.89	60	28
5	25	9	36.00	16	64.00	11	5
Average litter 2.56							
Av. litter weight 173.73g							
Totals	747	482		265		167	98
Percentages (mean) ..			69.52		35.47	63.01	36.98
V							
Alcoholic animals with treated great-grandparents and great-great-grandparents (F 3-4):							
1	19	16	84.21	3	15.78	2	1
2	70	56	80.00	14	20.00	9	5
3	120	88	73.33	36	26.66	16	16
4	68	32	47.05	36	52.94	21	15
5	10	7	70.00	3	30.00	2	1
Average litter 2.54							
Av. litter weight 175.88g							
Totals	287	199		88		50	38
Percentages (mean) ..			69.33		30.66	56.81	43.19
VI							
Alcoholic animals with treated great-great-grandparents (F 4):							
1	4	3	75.00	1	25.00	1	0
2	16	15	93.75	1	6.25	0	1
3	27	21	77.77	6	22.22	2	4
4	12	12	100.00	0		0	0
5	0	0		0		0	0
Average litter 2.46							
Av. litter weight 183.43g							
Totals	59	51		8		3	5
Percentages (mean) ..			86.45		13.54	37.50	62.50

"The records for the control show that the stock is strong and well. The average litter of the young is about three, and the average litter weight is 188.85 grams at birth. The data from 500 control individuals show a total mortality before reaching maturity of only 22.72 per cent. One familiar with breeding guinea pigs will recognize this as a very good record, far better than usual. In the last two columns of the table the total mortality has been divided into pre-natal and post-natal deaths. The absorbed embryos, premature births or absorptions, and stillborn young make up 53.91 per cent or about half of the total mortality. The post-natal mortality, under three months of age, was 46 per cent of the total. This equal division of pre-natal and post-natal mortality is the normal expectation for these animals. But Section II of the table shows that in the alcoholic generations the pre-natal is double the post-natal mortality, and among the F_1 animals in Section III the pre-natal mortality is more than two and a half times greater than the post-natal.

"Table XLVIII shows the size of litter in which the individuals occur. This is important, since animals born in large litters are at a disadvantage as compared with those born in small litters. The control section, for example, indicates that animals born in litters of two have a total mortality of only 17.5 per cent, while those born in litters of four suffer a mortality almost twice as high, or 33.3 per cent. The average litter size in the alcoholic group is smaller than among the control. This fact actually benefits their records and the mortality readings are corrected for the differences in litter size in the total mortality line of the table.

"Considering the successive generations it is seen that the offspring from directly treated parents present the poorest record of all. Almost 43 per cent of their young are lost before maturity, and actually 70.3 per cent of these are either ab-

sorbed as early embryos, aborted prematurely, or are stillborn. When these F_1 animals occur in small litters their chances for survival are greatly improved, so that of those occurring two in a litter only 28.5 per cent died, but of those in litters of four 73 per cent died. This fact conveys an idea of the great advantage possessed by members of the small litters. These are virtually partial litters the hardy survivors of an originally larger litter. When the records of the F_1 animals are corrected for litter size their mortality is about double that of the control.

" Many of these F_1 animals are defective and many are sterile; thus only the best of them are available to give rise to the following generations. Yet in spite of this partial elimination of the defective enough effect has been produced by the alcohol treatment on all individuals to cause the grandchildren to show an unfavorable record. When the records of the 747 animals of Section IV are compared with the control section the influence of the alcohol treatment is seen to be transmitted by the F_1 generation, although they had not been directly treated. The grandchildren of treated grandparents, the F_2 group, show a typical record closely comparable with that of the offspring from directly treated animals. The pre-natal mortality is here almost twice as high as the post-natal, and the total mortality is 64 per cent greater than among the control.

" Again, among the F_2 animals there are defective and sterile specimens. Thus another individual elimination and selection occurs in this generation and only the most vigorous individuals of the group are left to breed. These more vigorous specimens are in many cases mated with normal stock, so that the records of animals descended from treated great-grandparents are somewhat improved, yet the litter size is small and the mortality record for 287 such animals is 40 per cent higher than among the control.

" Finally, the constant elimination of the defective individu-

als through three generations and the matings with normal stock give animals descended from alcoholized great-great-grandparents which are superior in their records to the normal control animals. They are born in smaller litters than the control, but even when this is taken into account their mortality is actually only 64 per cent of that of the control animals. Such animals actually have a total mortality before the age of maturity of only 13.5 per cent.

"The fact that F_4 animals from alcoholic ancestry are superior in record to the control is a point of actual significance. There can be no doubt that the offspring from treated parents are decidedly inferior as compared with the control; and further, that their offspring, or the progeny from treated grandparents, are also evidently inferior, but during these generations an individual selection is taking place through the elimination from the race of the defective and sterile specimens. This selection finally brings out a group of unusually strong specimens from which all the weaklings have been eliminated, and although they are not quite so productive as the control, their offspring show a record superior in vitality.

"Should one desire to apply these experimental results to the human alcohol problem, it might be claimed that such elimination of unfit individuals had benefited the races of Europe, since all of the dominant races have a definitely alcoholic history, and the excessive use of alcohol was decidedly more general three or four generations ago than it is today. That is, certain families that were alcoholic three generations ago have not been excessively so in the more recent generations.

"When we consider the welfare of the race or stock rather than that of the individual it is found that the descendants of these groups of animals which suffered the highest mortalities and withstood the most vigorous selection are superior in quality to the descendants from the group less severely af-

fected. This individual selection furnishes a great advantage to the later generations as is shown by the superior quality of the F_4 group of guinea pigs in Table XLVIII."

It thus appears that when completely analyzed Stockard's long and painstaking experiments on guinea pigs are fully in accord with those of Pearl on fowls, *in respect of racially beneficial effect of the selection which accompanies the continued administration of alcohol.* Such differences as there are in the two cases in regard to the proportion of defective, but still in some degree viable individuals, probably are due mainly to two factors. One of these factors is probably a basic, inherent difference in response to alcohol of guinea pig protoplasm as compared with fowl protoplasm, at least in the strain of guinea pigs used by Stockard; the other is probably found in the fact that the guinea pig undergoes its embryonic development inside the body of the mother, and the fowl outside.

In neither of these recent papers of Stockard's, in which he has so strongly emphasized the racial benefit derived from the selective action of alcohol, does he give the slightest reference or hint that he is not the first investigator to show, or even to suggest, that alcohol had a selective action. The reader ignorant of the history of the subject is left to draw the conclusion that Stockard is the pioneer in showing a racially beneficial action of alcohol through selection, and just this conclusion has been repeatedly drawn by just such readers, during the last four years. But as has been set forth in this chapter, Pearl first presented experimental evidence for such a selective action in 1916, both in respect of germ cells and of developing embryos, for characters representing general vigor, followed by Danforth in 1919, for specific Mendelian unit characters.

Up to 1924 Stockard was the only investigator who had used guinea pigs in any critical or extensive study of the racial effect of alcohol. In that year Pictet (243) published a brief account

of some experiments in which male guinea pigs, starting at the age of one month, were subjected to alcohol daily, administered by inhalation over a period of 15 months. With other guinea pigs (one male and one female) the alcohol was started when they were 10 months of age and continued daily for a year. The young animals, alcoholized from the age of one month on, showed " a marked acceleration in growth, and as early as the end of the third month of the alcoholic régime exhibit an increase in weight considerably greater than that of the controls." This increase in growth took place mainly in the first 6 months, after which the curves became the same for the controls as for the alcoholized subjects. All the animals subjected to the alcohol régime remained vigorous and healthy. The fecundity of the alcoholized guinea pigs was perfectly normal. The average number of young per litter was 2.32 for the alcohol series, and 2.40 for the controls. The average birth weight of 84 young from alcoholic parentage was 85.60 grams against 82.90 for the controls, the average birth weight in Pictet's general stock being 75 grams. All the 84 offspring issuing from matings of alcoholized males with normal females, and from one mating in which both parents were alcoholized, were perfectly normal. Pictet emphasizes the fact that abnormal offspring *" absolument semblables à ceux que Stockard a signalés comme resultant de l'intoxication alcoholique,"* sometimes issue from matings of normal guinea pigs. He consequently advocates caution in ascribing monstrosities to the influence of alcohol, when using this form for experimental study. His experiments are less extensive than Stockard's, but the results are certainly more nearly in line with general experimental findings regarding the racial influence of alcohol in other animals.

An experimental study of the racial effect of continued administration of alcohol was begun in 1920 by Hanson (245,

246, 247, 248), using the albino rat as material. As in the work of Stockard, Pearl and Danforth, the alcohol was administered by inhalation. The characters so far published upon are chiefly body weight, body length and tail length. The work has been reported for four generations. The tentative conclusion so far reached is stated in the following words, after a résumé of the results of Pearl and Danforth (245, pp. 305–306).

"A germinal selection is probably also in progress in this work. This is evidenced by the fact that while in the early generations the amount of sterility was very great, at times endangering the continuance of the experiment, in the last two generations the fertility had been completely restored, and now equals, if not surpasses, that of the controls."

Hanson is cautious in drawing any conclusions and emphasizes the necessity of many successive generations. In regard to any influence of alcohol upon the sex ratio he finds that there is no such influence in the rat, confirming Pearl's conclusion to the same effect for the fowl, and shows by a proper biometric analysis of Stockard's data that it had no significant effect on the sex ratio in the guinea pig.

We must next consider the contributions of MacDowell (249–254). From the long list of this author's papers on alcohol I select, as in other cases, only the important original memoirs and summary articles for reference here. Their discussion has been deferred to this point, which brings the work slightly out of chronological order, because MacDowell has in the main dealt with an entirely different kind of character than any of the investigators previously mentioned. Indeed, the only prior experimental student of MacDowell's main problem was Arlitt (255, 256). MacDowell has concentrated upon the influence of alcohol (administered by inhalation) upon the *behavior* of white rats and their descendants. He has also

collected valuable information upon other characteristics, notably fertility and growth.

We may consider first the behavior results, which had to do with the acquirement of ability to go through a maze. The final conclusions of the last paper (253), which are consonant with and in part based upon the results of the first (249), are:

" White rats treated with maximum doses of alcohol fumes daily for 28 days before and on through training took more time per trial in running a circular maze than did their untreated brothers and sisters; this difference is significant when all the rats are averaged together. The same superiority of the normals is shown by each of the four strains separately; when each strain is divided according to sexes, most of the sets give similar results, but the small numbers introduce much irregularity into the curves.

" The criteria involving perfect trials give results that tend in the same direction as those given by the data on time, but they are less conclusive and definite.

" The treatment of the parents as well as the rats themselves causes no more modification in the maze behavior than was found in the rats originally treated, namely, a tendency to retard learning; but this tendency with the small numbers involved is either on or below the border-line of statistical significance.

" A small but consistent modification of the maze behavior of the untreated offspring from treated parents was found; this appeared more clearly in the criteria from perfect trials than in the time per trial.

" Seven rats from a normal pair did not appear to differ in their maze behavior from five rats from the same mother and a treated father.

" Untreated rats from parents treated with mild doses did

not show any difference in their maze behavior from their controls.

" Considering the evidence in this and the first paper in this series the following generalization is reached: Alcoholism in ancestors may modify the behavior of untreated descendants."

MacDowell's results respecting behavior are not particularly striking. Or to put the case the other way about, it would seem that the real net result of the large amount of laborious and careful experimental work which he has done on the point, is that alcoholization of ancestors produces no very definite or marked alteration in the behavior of progeny. What alteration he did get indicated a deleterious effect. But this is rather offset by Arlitt's (255) finding that the offspring of rats receiving 0.25 c.c. of alcohol per day, " were, as to intelligent behavior, generally on a par with, if not actually superior to, the offspring of normal rats."

It seems to me that the by-products of MacDowell's work, the data on fertility and growth, contribute rather more of value to the problem of the racial influence of alcohol than do the behavior results, chiefly because they are more definite.

Taking fertility first, Tables XLIX and L give MacDowell's data on litter size and litter number respectively (250, p. 126 and p. 131). His conclusions are:

" The treatment of white rats with alcohol by the inhalation method tends to reduce the size of litters. A reduction in the neighborhood of 10 per cent of the respective controls appears when the daily dose was small and when it was maximum, in the litters from treated rats as well as in rats whose parents or grandparents were treated. The numbers are not large enough to make this reduction statistically significant when each generation is considered by itself, but when all the generations are taken together, the difference between the test and control averages is significant.

TABLE XLIX

MacDowell's Data on Differences in Litter Size Between Alcoholized and Control White Rats

Treatment or Group of Animals	White Rats				Per cent Differences Controls 100%
	Litters		Differences − = tests less	Diff.	
	T	C		P. E.	
First Series					
Treated from untreated	13	13	− 1.08 ± 0.78	1.4	11.5
Second Series					
(1) Treated from untreated					
All litters	32	87	− 0.67 ± 0.34	1.9	10.0
1st litters	18	36	− 0.70 ± 0.41	1.7	10.2
"Balanced litters"	13	13	− 1.50 ± 0.56	2.6	20.0
(2) Treated from treated parents	29	14	− 0.66 ± 0.52	1.2	10.3
(3) Untreated from treated parents	20	15	− 0.70 ± 0.48	1.4	11.2
(4) Untreated from untreated parents and treated grandparents	25	19	− 0.88 ± 0.56	1.6	13.1
(1) and (2)	46	116	− 0.67 ± 0.28	2.4	10.0
(3) and (4)	45	34	− 0.81 ± 0.38	2.1	12.5
All generations	91	150	− 0.79 ± 0.22	3.6	11.9

"Given daily in maximum doses alcohol reduces the number of litters produced by the treated rats. Judging by the number of litters produced by their controls, this reduction is in the neighborhood of 65 per cent. The treatment of rats from treated parents made a smaller reduction (35 per cent) in the number of litters than when the treatment was confined to one generation. In contrast to this, rats whose parents or grandparents alone were treated produced from a third to a half as many again as the controls.

TABLE L

MacDowell's Data on Differences in Litter Number Obtained and Expected in White Rats

Generation	White Rats	
	% Difference between No. litters produced and No. normally expected	Diff. / P.E.
Second series		
Treated from untreated	− 64.86 ± 3.37	19.2
Treated from treated	− 35.45 ± 6.91	5.1
Untreated from treated	+ 33.33 ± 8.29	4.0
Untreated from untreated from treated	+ 55.60 ± 8.40	6.6

"These conclusions may be interpreted as due, first, to an inherited modification that reduces the size of litters, and second, to the selective elimination of germ-plasm bearing factors detrimental to litter production through the immediate reaction of alcohol upon the treated rats. Thus alcohol appears to modify fertility in two different ways, each working independently, and, in the generations after the first, in opposite directions.

"In so far as these results show a reduction in the total output, they agree with those of Stockard on guinea pigs and Pearl on fowl. Further than this the per cent reduction in litter size in the different generations is closely alike for rats and guinea pigs, while the results given by the number of litters agree with those of Pearl in showing immediate reduction in numbers, followed by a superiority in the survivors."

Regarding growth, MacDowell's data are presented in Table LI (from (251) p. 306, where the material is given in better form than in the original paper (252) devoted to this topic).

TABLE LI

THE WEIGHTS AT SUCCESSIVE AGES OF THE VARIOUS TYPES OF MALE TEST RATS COMPARED WITH THEIR RESPECTIVE CONTROLS

(From MacDowell)

Relation to Alcohol Treatment	Age in Days	Tests		Controls		Difference	*D/P.E.*
		Grams Averages	Nos.	Grams Averages	Nos.		
Treated from normal parents.....	40	70.43	70	72.95	64	+ 2.52 ± 1.40	1.8
	60	116.66	71	129.35	65	+ 12.69 ± 2.16	5.9
	90	149.63	61	176.63	55	+ 27.00 ± 3.16	8.5
	120	192.10	50	229.71	46	+ 37.61 ± 4.07	9.2
	150	215.39	38	257.97	39	+ 42.58 ± 4.81	8.8
	180	235.88	34	274.34	32	+ 38.46 ± 5.11	7.5
Treated from treated parents	40	83.86	15	81.81	16	− 2.05 ± 2.73	0.7
	60	128.00	15	131.81	16	+ 3.81 ± 3.81	1.0
	90	166.50	14	166.37	16	− 0.13 ± 4.96	0.0
	120	198.07	14	207.50	14	+ 9.43 ± 6.01	1.5
	150	228.33	12	236.50	14	+ 8.17 ± 6.48	1.2
	180	245.70	10	258.00	13	+ 12.30 ± 7.44	1.6
Untreated from treated parents.....	40	73.03	26	63.82	29	− 9.21 ± 2.37	3.8
	60	95.53	26	84.13	29	− 11.40 ± 2.56	4.4
	90	127.26	26	117.79	29	− 9.47 ± 3.64	2.6
	120	195.26	26	182.14	27	− 13.12 ± 5.13	2.5
	150	230.42	26	219.88	27	− 10.54 ± 6.24	1.6
	180	249.00	25	238.85	27	− 10.15 ± 7.61	1.3
Untreated from untreated parents and treated grandparents	40	75.90	10	80.57	7	+ 4.67 ± 5.08	0.9
	60	111.18	11	97.20	10	− 13.98 ± 5.35	2.6
	90	131.54	11	125.90	10	− 5.64 ± 11.64	0.5
	120	191.63	11	171.30	10	− 20.33 ± 16.66	1.2
	150	226.09	11	208.00	10	− 18.09 ± 16.20	1.1
	180	235.54	11	230.20	10	− 5.34 ± 13.52	0.4

Plus signs indicate the controls heavier; *D/P.E.* = difference divided by its probable error.

MacDowell points out that these results are very similar to those on fertility, and goes on to say:

" Just as the offspring of the treated rats appear to be genetically superior to the controls in the matter of litter production, so they are found to be superior in the matter of weight, with the result that when they themselves are treated, the immediate reducing effect of the alcohol makes them about equal somatically to their controls, instead of growing markedly slower as did their parents. This likeness in results leads to a similar interpretation for the weight as for the number of litters: the alcohol has acted as a selective agent, eliminating germinal material that included factors for slower growth."

We may now briefly consider the results of some of the experimental work on the subject with other animals as material. Nice (257–259) has published three papers reporting, among other things, the effect of parental alcoholism on the growth and activity of the progeny of white mice. Administering alcohol in the early experiments *per os* with food, and later by inhalation, his results on growth are summarized as follows: " Although the young of the alcohol mice when given alcohol themselves excelled all the other mice in growth, other young of these same mice when not given alcohol grew even faster." In other words the non-alcoholized progeny of alcoholized parents made the best growth records. Furthermore, Nice's data are such as to indicate that the result is due to a selective action of the alcohol.

Bluhm (260) performed experiments with white mice on the alteration of the normal sex ratio by the administration of alcohol to the male parent. A preliminary experiment on 195 complete normal litters containing 965 young gave a sex ratio of 79.36 ♂ : 100 ♀. Alcoholization was produced by injecting a solution of ethyl alcohol subcutaneously. Only male parents were injected. Sixty-seven alcoholic litters gave a sex ratio of

122.14 ♂ : 100 ♀. She concludes that this result may be explained on the assumption that there are two sorts of spermatozoa, male determiners and female determiners, and that the mortality of the latter sort is more diminished by the alcoholization than that of the former. The work seems carefully done, but should be repeated because so far out of line with other extensive experiments by Hanson, Stockard, and Pearl (*cf. supra*).

The most extensive and thorough experiments on the racial effects of alcohol with mice are those of Gyllenswärd (261). Alcohol was administered by inhalation. In the parental (alcoholized) and two offspring generations there were obtained altogether 1203 animals. No malformations of any sort were observed in any of them. There were no significant differences in the weights of alcohol and control offspring. The mortality in the treated parental generation was very slight. The period of gestation was normal, and the same in both treated and control groups. The percentage of infertile matings was from 2 to 5 per cent higher in the alcohol series than in the control. There was no evidence of damage to the germ plasm. The author states that the results agree well with those of Pearl, and are in opposition to those of Stockard.

Rost and Wolf (262) report the results of experiments on rabbits in which alcohol was introduced into the stomach by a tube. The general result was that no unfavorable effect of the alcohol upon the treated animals was observed, either in respect of fecundity, duration of pregnancy, weight, or general health. No animal which died and came to autopsy showed either cirrhosis or fatty degeneration of the liver, or inflammation of the mucous membrane of the stomach. Finally no deleterious effect upon the offspring of alcoholized parents was observed.

Bilski (263) experimented with the frog. The individuals

were alcoholized by immersions in solutions of alcohol of different strength, and for different lengths of time. The spawn of a female was divided and fertilized with the sperm of non-alcoholized males and of males alcoholized in varying degrees; and conversely the sperm cells of males were made to fertilize ova of females alcoholized in differing degrees. From the combination of single pairs and the comparison of their offspring, it was thought possible partly to eliminate the individual differences in the parents, so that the effect of the alcohol alone was left. His conclusions are that alcohol causes premature ovulation in the frog. No typical defects could be found in the offspring of even the most heavily alcoholized parents. More eggs developed from slightly alcoholized parents than from normal control parents. There was, however, usually an increased mortality in those broods, so that the number of offspring finally equalled or fell below the number in control broods. Bilski thinks that moderate alcoholization of parents has a stimulating effect upon eggs and sperm, and hence brings about the development of individuals from weak germ cells which normally would never start development. Later these individuals are eliminated by natural selection.

Pictet (244) has studied the effect of alcohol vapor on various species of *Lepidoptera,* namely *Vanessa urticae, V. io, Malaeosoma neustria,* and *Dendrolimus pini.* No degenerative effect upon the progeny was observed. Mann (264) found that a stable stock of the fruit fly *Drosophila melanogaster* " failed to show increased mutability or tendency to abnormality " when treated with ethyl alcohol fumes. She notes that alcohol appears to have some selective effect upon the germinal material, altering the expected Mendelian ratio.

Harrison (265) studied the effect of alcohol upon the offspring of the geometrid moth, *Selenia bilunaria.* His results are summarized as follows:

" (1) The offspring of treated parents neither included monstrosities nor displayed even slight abnormalities.

" (2) No new hereditable variations occurred.

" (3) The survivors of the treated batch grew, on the average, decidedly larger than their untreated relatives. This may have arisen simply from the elimination of the weaker individuals by the deleterious agent employed, or it may have been caused by some physiological action of the ethyl alcohol. In any case it agrees with Pearl's experience with his poultry; since his mortality rate was negligible, in all probability we have to look to both of the suggested causes for a correct explanation in this instance.

" (4) The progeny of the treated batches was greatly superior in several respects to that of the untreated controls.

" (5) The offspring from the cross between a treated male and an untreated female was superior to that of the reverse mating to much the same degree as the latter was better than the controls.

" (6) The superiority of the broods originating with treated parents was exhibited in:

" (a) The smaller percentage of embryos perishing in the shell, the exact figures being 2.01% and 2.9% against 7.8%.

" (b) The quicker development of the embryo.

" (c) The speedier rate of feeding up in the larva.

" (d) The lower rate of larva mortality, 0%, and 18% against 46%.

" (e) The greater mean weight of the male pupa.

" (f) The greater mean weight of the female pupa.

" (g) The quicker development of the imago and its earlier appearance.

" (h) Its richer pigmentation.

" (7) In sex ratio and in the percentage of fertile eggs no differences were perceptible.

" Except, therefore, in the percentage of fertile eggs and in characters only capable of manifestation in the lepidoptera, my data in the main tend to point in the same direction as those of Pearl.

" To explain the results I think we have to look to the cumulative effects of selection of two different types acting at different stages: (I) the first of these acts on the parental zygotes when the alcohol weeds out the weaker insects and preserves the stronger to perpetuate their race; (II) the second works directly on the germ cells of these selected, stronger, and sturdier survivors."

We now briefly summarize the whole case. It is to be noted that throughout this chapter I have followed the plan of directly quoting the several workers. This was done to forestall any possibility of the charge being made that I had read into their data interpretations different from their own.

I think that the following general conclusions may fairly be drawn as summarizing to date the important net results of the considerable body of careful painstaking research which had been done upon the problem.

1. The *racial* effect of alcohol is preponderantly either beneficial, or at the worst, not harmful. This is true for characters depending upon general vigor in guinea pigs (after early generations are passed), fowls, rats, mice, rabbits, insects, and probably frogs.

2. This beneficial racial effect appears to be the result primarily of the fact that alcohol acts as a definite, but not too drastic selective agent, both upon germ cells and developing embryos, eliminating the weak and leaving the strong.

3. The only racial effects of alcohol which can possibly be regarded as harmful which have yet been brought to light by this mass of experimental work are:

(a) The production of defective offspring in early genera-

tions by alcoholized guinea pigs (Stockard). This result is peculiar to the guinea pig, and is not confirmed, even for that animal, by Pictet. No such effect has been noted in any other animal: either the fowl (Pearl, Danforth); rat (MacDowell, Hanson); mouse (Gyllenswärd, Nice, Bluhm); rabbit (Rost and Wolf); frog (Bilski); or various insects (Pictet, Harrison, Mann).

(b) A possible slight reduction in activity and ability to learn of the offspring of white rats (MacDowell).

(c) A reduction in fertility following the administration of alcohol. But as this marks one element of a selective process which ultimately is beneficial to the race, it is questionable whether it should be reckoned a *racially* harmful effect at all.

Any application of these experimental results to man should be cautiously made, for the following reasons, among others:

The dosage of alcohol is in general much larger in the experimental work with animals than any consumption of alcohol by human beings.

The alcohol is administered in most of the experiments by inhalation, a process which has some very different physiological effects than drinking alcohol in solution.

The guinea pig case shows that some organisms may be quite differently affected racially, for a time at least, by alcohol, than are others. The conclusions drawn from one organism cannot safely be transferred to another in this case.

But regardless of any immediate application to man, it is an extremely satisfactory thing to have such a large body of critical experimental results, so consistently agreeing in regard to their main, broad conclusions. That much more work needs to be done in this field goes without saying, but the experimenter who begins now has a definite and consistent body of solidly grounded knowledge to start from, instead of the indefinite conjectures of fifteen years ago.

CHAPTER IX

SUMMARY AND CONCLUSIONS

THE results of the investigation which this book reports can be stated in much less time and space than the work itself required. They are:

1. In a fairly large and homogeneous sample of the working class population of Baltimore the moderate drinking of alcoholic beverages did not shorten life. On the contrary moderate steady drinkers exhibited somewhat lower rates of mortality, and greater expectation of life than did abstainers. This superiority is not great in the male moderate drinkers, and may not be significant statistically. But it certainly gives no support to the almost universal belief that alcohol always shortens life, even in moderate quantities.

2. Those persons in this experience who were heavy drinkers of alcoholic beverages exhibited considerably increased rates of mortality and diminished longevity, as compared with abstainers or moderate drinkers.

3. If both moderate drinkers and heavy drinkers in this sample of the population are pooled together, and the resulting heterogeneous group is compared with abstainers, the drinkers, as a class, have higher rates of mortality and lower expectation of life than the abstainers as a class. This result is in agreement with the experience of life insurance companies. But it is fully demonstrated in this book that this result appears only because the impaired heavy drinker risks are pooled with the actuarially superior moderate drinkers, and bring down the resulting pooled average.

4. Experiments by various workers, on such different forms of life as guinea pigs, fowls, rats, mice, rabbits, frogs and insects, agree in showing a beneficial effect of alcohol upon the race. This beneficial effect appears to be produced chiefly as a result of the remarkably sharp and precise selective action of this agent upon germ cells and developing embryos, killing off the weak and defective and leaving the strong and sound to survive and perpetuate the race. The prevalent notion that parental alcoholism tends to cause the production of weak, defective, or monstrous progeny is not supported by the extensive body of experimental work which has been done on the problem. Only one recent, critical experimenter has ever reported the production of defective offspring following parental alcoholism, and his results respecting this point are definitely *not* confirmed by another competent worker with the same animal, the guinea pig.

It seems clear, and entirely just, that anyone disagreeing with the conclusion reached in this study that there is no impairment of the life duration of moderate drinkers as compared with abstainers, must assume the burden of proof as to why the present considerable mass of objective data do not show a result opposite in sense to that which they do in fact show regarding this point. I am in no way constrained to explain why moderate drinkers and abstainers show similar life expectancies at all ages. I am content to rest upon the fact that it is so in the present statistics. On the principle of Occam's razor (*Entia non sunt multiplicanda praeter necessitatem*) the most probable explanation seems to me to be the simple one that the moderate consumption of alcoholic beverages has no deleterious biological effects. But since I have carefully refrained throughout the book from stating this as a *general* conclusion, I am in no wise obligated to prove it as such. Instead it is sufficient for the present merely to draw the specific

conclusion that in the considerable sample of the working class population of Baltimore here studied, moderate drinkers did live, on the average, just as long as total abstainers, and in truth a little longer.

These, then, are the results of this investigation. They seem to indicate, with great clearness, that any biological harmfulness chargeable against alcohol, in this group of over 5000 people, resulted solely from its abuse, and not from its reasonable and proper use. I said at the beginning of this book that the sole object of the study was to learn something about the purely biological effects of alcohol, as distinguished from its real or supposed social effects. Now that the work is finished I see no reason to change from this position. The social problem presented by alcohol seems to me to resolve itself finally into a matter of taste. The essential elements in the situation are these: (a) Alcohol when abused leads directly to more or less disastrous consequences; (b) some human beings are so constituted that they will abuse it, with greater or less frequency and regularity. Given this situation mankind divides itself promptly into two moieties, on the basis really of taste. Those in the one group feel it their most sacred duty to prevent the weak brother from getting the chance to liquidate his weakness in terms of this particular deleterious agent, at least. The other group feels that it is neither right nor decent to deprive the great bulk of normal humanity of a harmless source of pleasure in order that a small group of persons deficient in self control may theoretically be kept out of temptation.

The difference between these two groups of honest and sincere human beings is, I repeat, at bottom a matter of taste, of general outlook on life. There is no more hope of reconciling such a difference than there is of getting all men to agree that Beethoven's music is preferable to Irving Berlin's, or that Mohammedan dogma is more satisfying than Christian, or that

corned beef and cabbage is a more delectable dish than *confit d'oie*. To the resolution of such problems science can really contribute nothing effective. That elyng creature man is not a wholly rational animal in respect of his behavior. In fact he only acts rationally, if by natural endowment capable of doing so, when the consequences of failing so to act are immediately and sufficiently painful. Otherwise his emotions and tastes get full play. Up to the present time the consequences of minding other people's business relative to alcohol, on the one hand, or minding one's own business relative to this volatile compound, on the other hand, have in neither case been sufficiently painful to bring about anything approaching universally rational control of behavior in the premises.

Finally I must expressly disclaim any responsibility for the application of the results of this investigation to the business of individual human living. Whether any particular person chooses to be a teetotaler, a moderate drinker, or a sot, is a matter to be decided between himself, his inherited constitution, and "whatever gods may be." The only pertinent advice I can give him is that he learn, by precise experimentation, what constitutes moderate drinking *for him*. Nothing emerges more plainly from all the scientific work that has been done on alcohol, than that individual tolerance to alcohol is a highly variable phenomenon. Also it will be the part of wisdom to remember that a conclusion which is on the average true for a large statistical aggregate may not be so for a particular individual in that aggregate.

But why continue? Henry Arthur Jones (in *The English Review* for December, 1924) elucidated the matter profoundly and cheerfully:

"With respect to alcoholic disease, as with other diseases, the chief thing is to get oneself born with an immunity to it. I took this precaution against alcohol, and thereby have been

able to enjoy a bottle of wine a day for the last forty years, without ever getting drunk, without ever approaching drunkenness, without ever wishing to take more than was good for me. Such are the advantages of belonging to God's elect, as the apostle affectionately calls us. . . . As one born with a joyous capacity for taking a moderate, harmless quantity of wine, I strenuously protest against being degraded into equality with C_3 degenerates to whom alcohol is poison. Of all the devastating applications of the false and abominable doctrine of equality, this is one of the most intolerable. Why should a poor but healthy labouring man, whose rich brawny thews and vigorous stomach righteously demand a draught of refreshing ale, be condemned to meagre and gloomy equality with the rich valetudinarian, to whose queasy digestion a mere sip of alcohol is veritable poison? Away with this damnable doctrine of equality when it tells so hardly upon the working man! "

APPENDICES

APPENDIX I

THE GRADUATION OF THE MORTALITY EXPERIENCE

THE life table function used as the basis of the graduation throughout was the *death rate* at ages, 1000 q_x.

The logarithms of these death rates, calculated as described in Chapter IV, were fitted with cubic parabolae, by the method of least squares. The equations obtained were as follows:

Abstainer; Males	$y = 0.8558 + 0.0108x + 0.0112x^2 - 0.00020x^3$
Abstainer; Females	$y = 0.8447 - 0.0166x + 0.0173x^2 - 0.00057x^3$
All moderate; Males	$y = 0.8983 - 0.00059x + 0.0133x^2 - 0.00041x^3$
All moderate; Females	$y = 0.7178 - 0.0297x + 0.0210x^2 - 0.00076x^3$
Moderate occasional; Males..	$y = 0.9475 + 0.0042x + 0.0109x^2 - 0.00029x^3$
Moderate occasional; Females	$y = 0.7088 - 0.1411x + 0.0491x^2 - 0.0023x^3$
Moderate steady; Males	$y = 0.7778 - 0.0097x + 0.0150x^2 - 0.00042x^3$
Moderate steady; Females ..	$y = 0.6883 + 0.0525x + 0.0063x^2 - 0.000088x^3$
All heavy; Males	$y = 0.8795 + 0.1832x - 0.0145x^2 + 0.00063x^3$
Heavy occasional; Males	$y = 0.8912 - 0.0517x + 0.0243x^2 - 0.00081x^3$
Heavy steady; Males	$y = 0.7939 + 0.2197x - 0.0197x^2 + 0.00084x^3$

In these equations $y = \log_{10} 1000q_x$, and $x =$ age, measured in five year units from 25 years as origin. From these fitted q_x's complete life tables were calculated by the usual method for abstainers, all moderate, and all heavy, while for the other classes short life tables were calculated by Hayward's method (267, 268). These life tables all begin at 30 years of age to avoid the effect on the earlier age groups of the fact that many have not yet formed their permanent habits.

APPENDIX II

LIFE TABLES

In the following life tables the constants l_x, d_x, $1000q_x$, and $\mathring{e}_x$ are given annually for the four groups *Abstainer, All moderate, All heavy,* and *All drinkers,* in the case of males and for the two groups, *Abstainer* and *All moderate,* in the case of females. In calculating these tables all the available data were made use of and therefore the tables continued out to the bitter end of 110 years. It will be understood that from about age 90 on this amounts practically to a merely arithmetical procedure. The data are not sufficiently extensive at these higher ages to give reliable death rates, and there is probably exaggeration of centenarian ages, as is usual. In calculating the tables the assumption made by Glover was adopted that the death rates increase in geometrical progression at these higher ages to unity at age 115. This is biologically a dubious assumption, so far as concerns the upper limit of 115, but it seemed better to follow actuarial convention in this respect, rather than to arouse controversy on a point of very subsidiary importance.

TABLE I

Life Tables for Males, by Drinking Habits

Age	Abstainers				All moderate				All heavy				All drinkers			
	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	e_x	l_x	d_x	1000 q_x	$\mathring{e}_x$
30	100,000	754	7.54	36.34	100,000	814	8.14	36.75	100,000	1119	11.19	28.57	100,000	919	9.19	33.60
31	99,246	760	7.66	35.61	99,186	817	8.24	36.05	98,881	1188	12.01	27.89	99,081	956	9.65	32.91
32	98,486	768	7.80	34.88	98,369	822	8.36	35.34	97,693	1256	12.86	27.22	98,125	995	10.14	32.22
33	97,718	778	7.96	34.15	97,547	830	8.51	34.64	96,437	1325	13.74	26.57	97,130	1033	10.63	31.55
34	96,940	788	8.13	33.42	96,717	839	8.67	33.93	95,112	1393	14.65	25.93	96,097	1070	11.14	30.88
35	96,152	801	8.33	32.69	95,878	849	8.85	33.22	93,719	1461	15.59	25.31	95,027	1108	11.66	30.22
36	95,351	814	8.54	31.96	95,029	861	9.06	32.52	92,258	1528	16.56	24.70	93,919	1145	12.19	29.57
37	94,537	830	8.78	31.23	94,168	875	9.29	31.81	90,730	1592	17.55	24.11	92,774	1181	12.73	28.93
38	93,707	847	9.04	30.51	93,293	890	9.54	31.10	89,138	1655	18.57	23.53	91,593	1217	13.29	28.30
39	92,860	865	9.32	29.78	92,403	907	9.82	30.40	87,483	1716	19.61	22.97	90,376	1253	13.86	27.67
40	91,995	886	9.63	29.06	91,496	926	10.12	29.69	85,767	1773	20.67	22.41	89,123	1287	14.44	27.05
41	91,109	907	9.96	28.33	90,570	946	10.45	28.99	83,994	1828	21.76	21.88	87,836	1321	15.04	26.44
42	90,202	932	10.33	27.61	89,624	969	10.81	28.29	82,166	1879	22.87	21.35	86,515	1355	15.66	25.84
43	89,270	957	10.72	26.90	88,655	993	11.20	27.60	80,287	1926	23.99	20.84	85,160	1387	16.29	25.24
44	88,313	985	11.15	26.18	87,662	1020	11.63	26.90	78,361	1970	25.14	20.34	83,773	1419	16.94	24.65
45	87,328	1015	11.62	25.47	86,642	1048	12.09	26.21	76,391	2010	26.31	19.85	82,354	1450	17.61	24.07
46	86,313	1047	12.13	24.77	85,594	1078	12.59	25.53	74,381	2045	27.50	19.38	80,904	1481	18.30	23.49
47	85,266	1080	12.67	24.06	84,516	1109	13.12	24.85	72,336	2076	28.70	18.91	79,423	1511	19.02	22.92
48	84,186	1117	13.27	23.37	83,407	1144	13.71	24.17	70,260	2102	29.92	18.45	77,912	1539	19.76	22.35
49	83,069	1155	13.91	22.67	82,263	1179	14.33	23.50	68,158	2124	31.16	18.01	76,373	1567	20.52	21.79
50	81,914	1197	14.61	21.99	81,084	1217	15.01	22.84	66,034	2141	32.42	17.57	74,806	1594	21.31	21.24
51	80,717	1240	15.36	21.30	79,867	1257	15.74	22.18	63,893	2153	33.70	17.14	73,212	1621	22.13	20.69

TABLE I (*Continued*)

Age	Abstainers				All moderate				All heavy				All drinkers			
	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	e_x	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$
52	79,477	1286	16.18	20.63	78,610	1299	16.52	21.52	61,740	2161	35.00	16.72	71,591	1645	22.99	20.15
53	78,191	1335	17.07	19.96	77,311	1343	17.37	20.88	59,579	2164	36.32	16.31	69,946	1671	23.88	19.61
54	76,856	1386	18.03	19.30	75,968	1389	18.28	20.24	57,415	2162	37.66	15.91	68,275	1694	24.81	19.08
55	75,470	1439	19.07	18.64	74,579	1436	19.26	19.60	55,253	2156	39.02	15.51	66,581	1716	25.78	18.55
56	74,031	1495	20.20	18.00	73,143	1486	20.31	18.98	53,097	2146	40.41	15.12	64,865	1738	26.80	18.03
57	72,536	1554	21.43	17.36	71,657	1537	21.45	18.36	50,951	2131	41.83	14.73	63,127	1760	27.87	17.51
58	70,982	1616	22.77	16.73	70,120	1590	22.67	17.75	48,820	2113	43.28	14.36	61,367	1779	28.99	17.00
59	69,366	1680	24.22	16.10	68,530	1643	23.98	17.15	46,707	2091	44.76	13.98	59,588	1798	30.17	16.49
60	67,686	1746	25.79	15.49	66,887	1698	25.39	16.56	44,616	2064	46.27	13.61	57,790	1815	31.42	15.99
61	65,940	1814	27.51	14.89	65,189	1754	26.91	15.98	42,552	2035	47.83	13.25	55,975	1833	32.74	15.49
62	64,126	1883	29.37	14.30	63,435	1810	28.54	15.41	40,517	2002	49.42	12.89	54,142	1848	34.14	15.00
63	62,243	1954	31.40	13.71	61,625	1867	30.30	14.85	38,515	1967	51.07	12.54	52,294	1864	35.63	14.51
64	60,289	2027	33.62	13.14	59,758	1923	32.18	14.29	36,548	1929	52.77	12.18	50,430	1876	37.20	14.03
65	58,262	2099	36.03	12.58	57,835	1979	34.21	13.75	34,619	1888	54.53	11.83	48,554	1888	38.89	13.55
66	56,163	2171	38.66	12.03	55,856	2032	36.38	13.22	32,731	1845	56.36	11.49	46,666	1898	40.68	13.08
67	53,992	2242	41.53	11.50	53,824	2084	38.72	12.70	30,886	1799	58.26	11.14	44,768	1908	42.61	12.62
68	51,750	2312	44.67	10.97	51,740	2133	41.23	12.19	29,087	1752	60.25	10.80	42,860	1914	44.67	12.15
69	49,438	2378	48.10	10.46	49,607	2179	43.93	11.70	27,335	1704	62.33	10.46	40,946	1920	46.88	11.70
70	47,060	2440	51.85	9.97	47,428	2221	46.82	11.21	25,631	1654	64.52	10.13	39,026	1922	49.25	11.25
71	44,620	2496	55.95	9.48	45,207	2257	49.93	10.74	23,977	1602	66.81	9.79	37,104	1923	51.82	10.81
72	42,124	2546	60.44	9.01	42,950	2288	53.26	10.28	22,375	1549	69.23	9.45	35,181	1920	54.58	10.37
73	39,578	2586	65.35	8.56	40,662	2311	56.83	9.83	20,826	1495	71.78	9.12	33,261	1915	57.57	9.94
74	36,992	2617	70.74	8.13	38,351	2326	60.65	9.39	19,331	1440	74.49	8.79	31,346	1906	60.82	9.52

TABLE I (*Continued*)

Age	Abstainers				All moderate				All heavy				All drinkers			
	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$
75	34,375	2635	76.65	7.71	36,025	2333	64.75	8.96	17,891	1384	77.35	8.45	29,440	1894	64.34	9.10
76	31,740	2639	83.13	7.31	33,692	2329	69.14	8.55	16,507	1327	80.40	8.12	27,546	1878	68.17	8.69
77	29,101	2626	90.25	6.92	31,363	2316	73.83	8.15	15,180	1270	83.64	7.79	25,668	1857	72.34	8.29
78	26,475	2596	98.07	6.56	29,047	2290	78.85	7.76	13,910	1212	87.11	7.45	23,811	1831	76.90	7.90
79	23,879	2547	106.66	6.22	26,757	2253	84.22	7.38	12,698	1153	90.81	7.12	21,980	1800	81.88	7.52
80	21,332	2477	116.10	5.90	24,504	2204	89.95	7.01	11,545	1094	94.79	6.78	20,180	1763	87.35	7.14
81	18,855	2328	123.47	5.61	22,300	2149	96.36	6.65	10,451	1060	101.39	6.43	18,417	1724	93.65	6.78
82	16,527	2170	131.31	5.33	20,151	2080	103.22	6.31	9,391	1018	108.45	6.10	16,693	1676	100.40	6.43
83	14,357	2005	139.64	5.06	18,071	1998	110.57	5.98	8,373	971	116.00	5.78	15,017	1617	107.64	6.09
84	12,352	1834	148.50	4.80	16,073	1904	118.45	5.66	7,402	918	124.08	5.48	13,400	1546	115.41	5.76
85	10,518	1661	157.92	4.55	14,169	1798	126.89	5.35	6,484	861	132.72	5.18	11,854	1467	123.73	5.45
86	8,857	1487	167.94	4.31	12,371	1682	135.93	5.06	5,623	798	141.96	4.90	10,387	1378	132.66	5.15
87	7,370	1316	178.60	4.08	10,689	1556	145.61	4.77	4,825	733	151.84	4.63	9,009	1281	142.23	4.86
88	6,054	1150	189.93	3.86	9,133	1425	155.98	4.50	4,092	665	162.42	4.37	7,728	1179	152.49	4.58
89	4,904	991	201.99	3.64	7,708	1288	167.09	4.24	3,427	595	173.73	4.12	6,549	1070	163.49	4.31
90	3,913	841	214.80	3.44	6,420	1149	179.00	3.99	2,832	526	185.83	3.88	5,479	961	175.28	4.06
91	3,072	702	228.43	3.25	5,271	1011	191.75	3.75	2,306	458	198.77	3.65	4,518	849	187.93	3.81
92	2,370	576	242.92	3.06	4,260	875	205.41	3.52	1,848	393	212.61	3.43	3,669	739	201.48	3.58
93	1,794	463	258.34	2.88	3,385	745	220.04	3.31	1,455	331	227.41	3.22	2,930	633	216.02	3.36
94	1,331	366	274.73	2.71	2,640	622	235.72	3.10	1,124	273	243.25	3.02	2,297	532	231.60	3.15
95	965	282	292.17	2.54	2,018	510	252.51	2.90	851	222	260.19	2.83	1,765	438	248.31	2.94
96	683	212	310.71	2.39	1,508	408	270.50	2.71	629	175	278.30	2.64	1,327	353	266.22	2.75
97	471	156	330.42	2.24	1,100	319	289.77	2.53	454	135	297.69	2.47	974	278	285.42	2.57

TABLE I (*Concluded*)

Age	Abstainers				All moderate				All heavy				All drinkers			
	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$
98	315	111	351.38	2.09	781	242	310.41	2.36	319	102	318.41	2.30	696	213	306.01	2.39
99	204	76	373.68	1.96	539	179	332.52	2.20	217	74	340.59	2.15	483	159	328.09	2.22
100	128	51	397.39	1.83	360	128	356.21	2.04	143	52	364.30	2.00	324	114	351.75	2.07
101	77	32	422.61	1.71	232	89	381.59	1.89	91	35	389.67	1.86	210	79	377.13	1.92
102	45	20	449.43	1.59	143	58	408.77	1.76	56	23	416.81	1.72	131	53	404.33	1.77
103	25	12	477.95	1.48	85	37	437.90	1.62	33	15	445.83	1.59	78	34	433.50	1.64
104	13	7	508.26	1.37	48	23	469.08	1.50	18	9	476.88	1.47	44	20	464.77	1.51
105	6	3	540.52	1.27	25	13	502.50	1.38	9	5	510.08	1.36	24	12	498.30	1.39
106	3	2	574.82	1.17	12	6	538.31	1.27	4	2	545.61	1.25	12	6	534.26	1.28
107	1	1	611.29	1.08	6	3	576.65	1.16	2	1	583.59	1.15	6	4	572.80	1.17
108	..	..	.....	....	3	2	617.73	1.06	1	1	624.24	1.05	2	1	614.12	1.06
109	..	..	.....	....	1	1	661.73	0.96	..	..	.....	....	1	1	658.42	0.96

TABLE II

Life Tables for Females, by Drinking Habits

Age	Abstainers				All moderates			
	l_x	d_x	1000 q_x	$\mathring{e}_x$	l_x	d_x	1000 q_x	$\mathring{e}_x$
30	100,000	700	7.00	37.63	100,000	511	5.11	40.95
31	99,300	701	7.06	36.89	99,489	511	5.14	40.16
32	98,599	704	7.14	36.15	98,978	514	5.19	39.37
33	97,895	710	7.25	35.40	98,464	518	5.26	38.57
34	97,185	716	7.37	34.66	97,946	523	5.34	37.77
35	96,469	725	7.52	33.91	97,423	531	5.45	36.97
36	95,744	736	7.69	33.16	96,892	540	5.57	36.17
37	95,008	749	7.88	32.42	96,352	550	5.71	35.37
38	94,259	763	8.10	31.67	95,802	563	5.88	34.57
39	93,496	781	8.35	30.93	95,239	577	6.06	33.77
40	92,715	799	8.62	30.18	94,662	594	6.27	32.97
41	91,916	820	8.92	29.44	94,068	611	6.50	32.18
42	91,096	843	9.25	28.70	93,457	634	6.78	31.39
43	90,253	867	9.61	27.96	92,823	653	7.04	30.60
44	89,386	895	10.01	27.23	92,170	677	7.35	29.81
45	88,491	924	10.44	26.50	91,493	704	7.69	29.03
46	87,567	956	10.92	25.77	90,789	733	8.07	28.25
47	86,611	991	11.44	25.05	90,056	764	8.48	27.47
48	85,620	1027	12.00	24.34	89,292	798	8.94	26.71
49	84,593	1067	12.61	23.63	88,494	834	9.43	25.94
50	83,526	1109	13.28	22.92	87,660	874	9.97	25.18
51	82,417	1155	14.01	22.22	86,786	916	10.56	24.43
52	81,262	1203	14.80	21.53	85,870	963	11.21	23.69
53	80,059	1253	15.65	20.85	84,907	1011	11.91	22.95
54	78,806	1307	16.58	20.17	83,896	1064	12.68	22.22
55	77,499	1363	17.59	19.50	82,832	1119	13.51	21.50
56	76,136	1422	18.68	18.85	81,713	1178	14.42	20.79
57	74,714	1484	19.86	18.19	80,535	1241	15.41	20.08
58	73,230	1549	21.15	17.55	79,294	1308	16.49	19.39
59	71,681	1616	22.54	16.92	77,986	1377	17.66	18.71
60	70,065	1685	24.05	16.30	76,609	1450	18.93	18.04
61	68,380	1757	25.69	15.69	75,159	1527	20.32	17.37
62	66,623	1829	27.46	15.09	73,632	1607	21.82	16.72
63	64,794	1904	29.38	14.50	72,025	1689	23.45	16.09
64	62,890	1979	31.46	13.93	70,336	1774	25.22	15.46

TABLE II (*Continued*)

Age	Abstainers				All moderates			
	l_x	d_x	1000 q_x	e_x	l_x	d_x	1000 q_x	$\mathring{e}_x$
65	60,911	2053	33.71	13.36	68,562	1861	27.15	14.85
66	58,858	2127	36.14	12.81	66,701	1950	29.23	14.25
67	56,731	2199	38.77	12.27	64,751	2039	31.49	13.66
68	54,532	2270	41.62	11.75	62,712	2128	33.93	13.09
69	52,262	2336	44.70	11.24	60,584	2216	36.57	12.53
70	49,926	2397	48.02	10.74	58,368	2301	39.42	11.99
71	47,529	2453	51.61	10.25	56,067	2383	42.50	11.46
72	45,076	2501	55.49	9.78	53,684	2460	45.82	10.95
73	42,575	2540	59.66	9.33	51,224	2530	49.39	10.45
74	40,035	2569	64.17	8.89	48,694	2592	53.24	9.96
75	37,466	2586	69.01	8.47	46,102	2644	57.36	9.50
76	34,880	2589	74.23	8.06	43,458	2685	61.79	9.04
77	32,291	2578	79.83	7.66	40,773	2713	66.53	8.61
78	29,713	2551	85.86	7.28	38,060	2725	71.60	8.18
79	27,162	2508	92.32	6.92	35,335	2722	77.02	7.78
80	24,654	2447	99.24	6.57	32,613	2700	82.78	7.38
81	22,207	2354	106.01	6.24	29,913	2659	88.89	7.01
82	19,853	2248	113.25	5.92	27,254	2601	95.44	6.64
83	17,605	2130	120.97	5.62	24,653	2527	102.49	6.29
84	15,475	2000	129.23	5.32	22,126	2435	110.05	5.95
85	13,475	1860	138.04	5.04	19,691	2327	118.17	5.62
86	11,615	1713	147.47	4.76	17,364	2203	126.89	5.31
87	9,902	1560	157.53	4.50	15,161	2066	136.25	5.01
88	8,342	1404	168.28	4.25	13,095	1916	146.30	4.72
89	6,938	1247	179.75	4.01	11,179	1756	157.10	4.44
90	5,691	1093	192.02	3.77	9,423	1590	168.69	4.18
91	4,598	943	205.13	3.55	7,833	1419	181.13	3.93
92	3,655	801	219.12	3.34	6,414	1248	194.50	3.68
93	2,854	668	234.07	3.14	5,166	1079	208.85	3.45
94	2,186	547	250.04	2.94	4,087	917	224.26	3.23
95	1,639	438	267.10	2.76	3,170	763	240.81	3.02
96	1,201	343	285.33	2.58	2,407	622	258.58	2.82
97	858	261	304.80	2.42	1,785	496	277.65	2.63
98	597	195	325.60	2.26	1,289	384	298.14	2.45
99	402	140	347.81	2.10	905	290	320.14	2.28
100	262	97	371.54	1.96	615	211	343.76	2.11
101	165	66	396.90	1.82	404	149	369.12	1.96

TABLE II (*Concluded*)

Age	Abstainers				All moderates			
	l_x	d_x	1000 q_x	e_x	l_x	d_x	1000 q_x	$\mathring{e}_x$
102	99	42	423.97	1.69	255	101	396.36	1.81
103	57	26	452.91	1.57	154	66	425.60	1.67
104	31	15	483.80	1.45	88	40	457.00	1.54
105	16	8	516.82	1.34	48	24	490.72	1.42
106	8	4	552.09	1.23	24	13	526.93	1.30
107	4	3	589.75	1.13	11	6	565.81	1.19
108	1	1	630.00	1.03	5	3	607.55	1.08
109	..	..	.....	....	2	1	652.38	0.98
110	..	..	.....	....	1	1	700.52	0.88

LITERATURE CITED

The references to the literature here given are only those cited in the text. They comprise a selection from an extensive annotated bibliography on the biological effects of alcohol, which may be published later as a separate work.

1. Pearl, R., and Parker, S. L. Experimental studies on the duration of life. I. Introductory discussion of duration of life in *Drosophila.* Amer. Nat. Vol. 55, pp. 481–509, 1921.
2. Pearl, R. A note on the inheritance of duration of life in man. Amer. Jour. Hyg. Vol. 2, pp. 229–233, 1922.
3. Pearl, R., and Parker, S. L. Experimental studies on the duration of life. II. Hereditary differences in duration of life of line-bred strains of *Drosophila.* Amer. Nat. Vol. 56, pp. 174–187, 1922.
4. *Id.* Experimental studies, etc. III. The effect of successive etherizations on the duration of life of *Drosophila. Ibid.,* pp. 273–280, 1922.
5. *Id.* Experimental studies, etc. IV. Data on the influence of density of population on duration of life in *Drosophila. Ibid.,* pp. 312–321, 1922.
6. *Id.* Experimental studies, etc. V. On the influence of certain environmental factors on duration of life in *Drosophila. Ibid.,* pp. 385–398, 1922.
7. Pearl, R. Experimental studies, etc. VI. A comparison of the laws of mortality in *Drosophila* and in man. *Ibid.,* pp. 398–405, 1922.
8. *Id.* The Biology of Death. Philadelphia and London (J. B. Lippincott Co.), 1922. 275 pp.
9. Pearl, R., and Parker, S. L. New experimental data on the influence of density of population upon duration of life in *Drosophila.* Amer. Jour. Hyg., Vol. 3, pp. 94–97, 1923.

10. Pearl, R., and Doering, C. R. A comparison of the mortality of certain lower organisms with that of man. Science, N. S., Vol. 57, pp. 209–212, 1923.
11. Pearl, R., Parker, S. L., and Gonzalez, B. M. Experimental studies, etc. VII. The Mendelian inheritance of duration of life in crosses of wild type and quintuple stocks of *Drosophila melanogaster*. Amer. Nat., Vol. 57, pp. 153–192, 1923.
12. Pearl, R. Interrelations of the biometric and experimental methods of acquiring knowledge; with special reference to the problem of duration of life. The Harvey Society Lectures, 1921–1922, pp. 179–205, 1923.
13. *Id.* Duration of life as an index of constitutional fitness. Poultry Science, Vol. 3, pp. 1–10, 1923.
14. Gonzalez, B. M. Experimental studies, etc. VIII. The influence upon duration of life of certain mutant genes of *Drosophila melanogaster*. Amer. Nat., Vol. 57, pp. 289–325, 1923.
15. Pearl, R., and Parker, S. L. Experimental studies, etc. IX. New life tables for *Drosophila*. *Ibid.* Vol. 58, pp. 71–82, 1924.
16. *Id.* Experimental studies, etc. X. The duration of life of *Drosophila melanogaster* in the complete absence of food. *Ibid.*, Vol. 58, pp. 193–218, 1924.
17. Pearl, R. Starvation life curves. Nature, Vol. 113, p. 854, 1924.
18. *Id.* A note on the inheritance of duration of life in man. Amer. Jour. Hyg., Vol. 2, pp. 229–233, 1922.
19. *Id.* The age at death of the parents of the tuberculous and the cancerous. *Ibid.*, Vol. 3, pp. 71–89, 1923.
20. *Id.* Preliminary account of an investigation of factors influencing longevity. Journal of the American Medical Association, Vol. 82, pp. 259–264, 1924.
21. *Id.* Studies in Human Biology. Baltimore (Williams and Wilkins), 1924.
22. Rosenfeld, G. Der Alkohol als Nährungsmittel. Zentralbl. f. innere Med. Bd. 27, pp. 289–299, 1906.
23. Mendel, L. B., and Hilditch, W. The influence of alcohol upon nitrogenous metabolism in men and animals. Amer. Jour. Physiol., Vol. 27, pp. 1–23, 1910.
24. Dodge, R., and Benedict, F. G. Physiological Effects of Alcohol. Carnegie Inst. of Washington Publ., No. 232, 1915.

25. Moore, B., and Webster, T. A. Studies of photosynthesis in freshwater algae. Proc. Roy. Soc., Series B, Vol. 91, pp. 201–215, 1919–20.
26. Bills, C. E. Some effects of the lower alcohols on *Paramecium*. Biol. Bulletin, Vol. 47, pp. 253–264, 1924.
27. Camus, L. Influence du régime alimentaire sur la toxicité de l'absinthe et de l'alcool. C. R. Soc. Biol. de Paris, T. 61, pp. 333–336, 1906.
28. Kochmann, M. Einfluss des Alkohols auf den hungernden Organismus. München. Med. Wchnschr. Bd. 56, pp. 549–551, 1909.
29. Alcohol: its Action on the Human Organism. (Second edition, revised.) Prepared by the Alcohol Investigation Committee of the Medical Research Council. Published by H. M. Stationery Office, 1924.
30. Starling, E. H., *et al.* The Action of Alcohol on Man. London (Longmans, Green and Co.), 1923.
31. Friedenwald, J. The comparative toxicity of various alcoholic beverages. Trans. Assoc. Amer. Physicians, Philadelphia, Vol. 26, pp. 60–66, 1911.
32. Whitney, D. D. The poisonous effects of alcoholic beverages not proportional to their alcoholic contents. Science, N. S. Vol. 33, pp. 587–590, 1911.
33. Yves-Guyot. La question de l'alcool. Allégations et réalités. Paris (Alcan), 1917.
34. Pearl, R. The experimental modification of germ cells. I. General plan of experiments with ethyl alcohol and certain related substances. Jour. Exper. Zool., Vol. 22, pp. 125–164, 1917.
35. *Id.* II. The effect upon the domestic fowl of the daily inhalation of ethyl alcohol and certain related substances. *Ibid.*, pp. 165–186, 1917.
36. *Id.* III. The effect of parental alcoholism, and certain other drug intoxications, upon the progeny. *Ibid.*, pp. 241–310, 1917.
37. *Id.* Some effects of the continued administration of alcohol to the domestic fowl, with special reference to the progeny. Proc. Nat. Acad. Sci., Vol. 2, pp. 675–683, 1916.
38. Völtz, W., and Baudrexel, A. Ueber die vom tierischen Organismus unter verschiedenen Bedingungen ausgeschiedenen Alkoholmengen. II. Mitth. Pflüger's Arch. Bd. 142, pp. 47–88, 1911. See also other papers in the same series.

39. Völtz, W., Förster, R., and Baudrexel, A. Ueber die Verwertung des Bierextraktes und des Bieres im menschlichen und tierischen Organismus. *Ibid.* Bd. 134, pp. 133–258, 1910.

40. Stockard, C. R., and Papanicolaou, G. N. Further studies on the modification of the germ-cells in mammals; the effect of alcohol on treated guinea pigs and their descendants. Jour. Exper. Zool. Vol. 26, pp. 119–226, 1918. (In this paper references will be found to Stockard's earlier work.)

41. Loewy, A., and von der Heide, R. Ueber die Giftwirkung von Methyl- und Aethylalkohol bei ihrer Einatmung. Berliner klin. Wchnschr. Bd. 55, pp. 67–69, 1918.

42. Carpenter, T. M., and Babcock, E. B. The concentration of alcohol in the tissues of hens after inhalation. Amer. Jour. Physiol., Vol. 49, pp. 128–129, 1919–1920.

43. Heron, D. A Second Study of Extreme Alcoholism in Adults, with Special Reference to the Home Office Inebriate Reformatory Data. Eugenics Lab. Mem. XVII, pp. 1–95, 1912.

44. Branthwaite, R. E. Annual Report for 1909 of the Inspector under the Inebriates Act. H. M. Stat. Off. Cd. 5799, pp. 55–93, 1911.

45. Welch, W. H. The Pathological Effects of Alcohol. *In* "Physiological Aspects of the Liquor Problem." Boston and New York, 1903, Vol. II, pp. 349–374.

46. Formad, H. F. The "pig-backed" or alcoholic kidney of drunkards. A contribution to the post-mortem diagnosis of alcoholism. Trans. Assoc. Amer. Physicians, Vol. 1, pp. 225–236, 1886.

47. Moore, R. M. On the comparative mortality among assured lives of abstainers and non-abstainers from alcoholic beverages. Jour. Inst. Act., Vol. 38, pp. 213–276, 1904.

48. Foudray, E. United States Abridged Life Tables, 1919–1920. Washington (Bureau of the Census), 1923.

49. Pearl, R. Alcohol and mortality. In Starling's "The Action of Alcohol on Man," pp. 213–286, 1923. (See (30)).

50. Reed, R. Facts versus fanaticism: A review of 200 nervous and mental cases tending to show the negligible importance of alcohol as an essential factor in their causation. Lancet — Clinic (Cincinnati), Vol. 112, pp. 421–427, 1914.

51. Hultgen, J. F. 406 cases of alcoholism, consecutive individual

observations. A clinical study accompanied by comparative statistics. Preliminary report. Jour. Inebriety, Vol. 31, pp. 117–123, 1909.

52. Etchepare, B. El alcoholismo mental en el Uruguay. Rev. med. d. Uruguay, T. 12, pp. 117–148, 1909.
53. Minor, L. S. Statistische Angaben und Beobachtungen aus den Gebiet des Alkoholismus. Russ. med. Rundschau, Bd. 9, pp. 189–198, 225–233, 1911.
54. Petro, F. La pazzia alcoolia in provincia di Cuneo. Gior. d. r. Soc. ital. d'ig., T. 33, pp. 251–264, 289–301, 337–352, 1911.
55. Lombroso, G. L'alcoolismo in Italia. Arch. di antrop. crim. Ser. 4, T. 8, pp. 449–461, 1916.
56. Kauffmann, M. Die Uebertreibungen der Abstinenz. Reichs. Med.-Ang. Bd. 36, pp. 289–291, 1911.
57. Mercier, C. Inaugural address on drunkenness and the physiological effects of alcohol. Lancet, 1912 (Vol. II), pp. 1492–1496, 1912.
58. Rogers, A. M. Some observations during 18 years' experience with drug and liquor habitués. Wisconsin Med. Jour., Vol. 12, pp. 40–43, 1913–14.
59. Williams, E. H. The liquor question in medicine. Med. Rec., Vol. 85, pp. 612–614, 1914.
60. Sumner, W. G. Folkways. Boston (Ginn and Co.). No date of publication anywhere stated. The book was copyrighted by the author, according to a statement on the reverse of the title page, in 1907.
61. Becker, H. Zur Toxikologie des Alkohols. Klin-therap. Wchnschr. Bd. 27, pp. 305–313, 1920.
62. Cooper, J. W. Astley. Pathological Inebriety. Its Causation and Treatment. London, 1913.
63. Mott, F. W. Alcohol and insanity. Brit. Jour. Inebriety, Vol. 9, pp. 5–27, 1911.
64. Maupaté, L. et Nollens, C. Contribution à l'étude de l'alcoolisme chez la femme dans le nord. Echo méd. du nord (Lille), T. 11, pp. 277–282, 1907.
65. Yule, G. U. An Introduction to the Theory of Statistics. Seventh edition. London (Griffin), 1924.
66. Pearl, R. Introduction to Medical Biometry and Statistics. Philadelphia (W. B. Saunders Co.), 1923.

67. Kirby, G. H. Race and alcoholic insanity. Jour. Amer. Med. Assoc. Vol. 57, pp. 9–11, 1911.
68. Cadbury, W. W. Idiosyncrasy to alcohol. Jour. Amer. Med. Assoc. Vol. 73, pp. 1935–1936, 1919.
69. Timbs, J. English Eccentrics and Eccentricities. A New Edition. London (Chatto and Windus), 1877.
70. Kabrel, G. Ueber die Bedeutung kleiner Alkoholdosen. Hyg. Rundschau, Bd. 19, pp. 577–586, 1909.
71. Becker, W. H. Eine Maximaldosis des Alkohols. Therap. Monatsch. Bd. 22, pp. 444–458, 1908.
72. Kelly, H. A. What is intoxication? Baltimore Evening Sun, Nov. 26, 1924.
73. Charpentier, A. Query drunk? Lancet, Oct. 25, 1924, pp. 885–886.
74. Glover, J. W. United States Life Tables 1890, 1901, 1910, and 1901–1910. Washington (Bureau of the Census), 1921.
75. Pearl, R. Alcohol and life duration. Brit. Med. Jour., May 31, 1924, pp. 948–950.
76. *Id.* The influence of alcohol on duration of life. Proc. Nat. Acad. Sci., Vol. 10, pp. 231–237, 1924.
77. Henderson, R. Mortality Laws and Statistics. New York, 1915, pp. v + 111.
78. Howard, W. T. Public Health Administration and the Natural History of Disease in Baltimore, Maryland, 1797–1920. Washington (Carnegie Institution Publ. No. 351), 1924, pp. vi + 565.
79. Tiffany, W. H. Prize Essay on the Relations of Temperance and Intemperance in Life Insurance. 8vo (n.p.: n.d.), 15 pp.
80. Burns, D. Vital statistics in relation to the use of intoxicating liquors. Tr. Nat. Assoc. Promot. Social Sci., 1864. London, pp. 539–545, 1865.
81. Wey, W. C. Additional thoughts concerning inebriety and life insurance. Med. Rev. N. Y., Vol. 10, pp. 49–51, 1875.
82. Drysdale, C. R. Influence de l'alcool sur la durée de la vie. Rev. scient. Paris, T. 40, pp. 429–432, 1887.
83. Bentham, R. The comparative death-rate of total abstainers and moderate drinkers. Lancet, Vol. 2, p. 997, 1890.
84. Whyte, J. Alcohol and longevity. Med. Pioneer. London, Vol. 4, pp. 92–95, 1895–1896.
85. Webster, G. W. The prevalence of alcoholism and its influence on

mortality and morbidity. Quart. J. Inebr. Hartford, Vol. 21, pp. 234–253, 1899. Also in Illinois M. J., Vol. 49, pp. 355–364, 1899–1900.

86. Hoppe. Alcoholismus und Lebensversicherung. Alcoholismus, Dresden. Bd. 1, pp. 268–272, 1900.
87. Sullivan, W. C. Inebriety and suicide. Brit. Jour. Inebriety, Vol. 1, pp. 25–28, 1903–04.
88. Westergaard, H. Was lehrt die Statistik in Betreff des Einflusses der geistigen Getränke auf die Gesundheit? Alkoholfrage, Dresden, Bd. 1, pp. 227–238, 1904.
89. Whittaker, T. P. Alcoholic beverages and longevity. Contemp. Rev. Vol. 85, pp. 413–429, 1904.
90. Benedict, F. G. Scientific aspects of moderate drinking. Quart. Jour. Inebriety, Hartford, Vol. 27, pp. 1–22, 1905.
91. Eccles, W. McA. Alcohol and life insurance. In The Drink Problem. London, 1907, pp. 152–160.
92. *Id.* The relation of alcohol to physical deterioration and national efficiency. Jour. Inebriety, Boston, Vol. 30, pp. 67–83, 1908.
93. Kress, D. H. Disease and mortality from alcohol. *Ibid.* Vol. 30, pp. 103–106, 1908.
94. Stille. Alcohol und Lebensdauer: eine Entgegnung. Med. Klin. Berlin, Bd. 4, pp. 1759–60, 1908.
95. Knight, H. W. The relation of alcohol to hygiene. St. Louis Med. Rev. N. S. Vol. 5, pp. 238–240, 1911.
96. McMahon, T. F. The use of alcohol and life insurance risks. Med. Rec., Vol. 80, pp. 1121–1123, 1911.
97. von Engelhardt. Die Alkoholfrage in individuel- und sozial-hygienischer Beleuchtung. St. Petersburg Med. Ztschr. Bd. 37, pp. 316–320, 1912.
98. Hunter, A. Effect of alcohol on longevity. Amer. Med. N. S. Vol. 9, pp. 106–108, 1914.
99. Lorentzen, G. Ueber die Schäden und Bekämpfung des Alkoholmissbrauchs vom Standpunkte der öffentlichen Gesundheit. Deutsch. vrtljschr. f. öff. Gsndhtspflg. Bd. 46, pp. 470–517, 1914.
100. Hunter, A. Effect of alcohol on longevity. Med. Rev. of Rev. Vol. 21, pp. 16–25, 1915. (Same material as No. 98.)
101. Fisk, E. L. Alcohol and life insurance. Atlantic Monthly, Vol. 118, pp. 624–634, 1916.

102. *Id.* Alcohol and human efficiency. *Ibid.* Vol. 119, pp. 43–50, 1917.
103. *Id.* Alcohol and physiology. *Ibid.* Vol. 119, pp. 203–210, 1917.
104. Hunter, A. Life insurance and drinking habits. Proc. Nat. Conf. Char. Chicago, Vol. 43, pp. 80–93, 1916.
105. Newsholme, A. The social aspects of the alcohol problem. Practitioner, Vol. 113, pp. 216–225, 1924.
106. Powell, R. D. Alcohol in its relation to life assurance. *Ibid.* Vol. 113, pp. 264–269, 1924.
107. Crothers, T. D. How far does the moderate use of liquor affect longevity? Med. Exam. and Pract., N. Y., Vol. 14, pp. 409–411, 1904.
108. *Id.* The effects of alcohol and tobacco on life expectancy. N. Y. Med. J., Vol. 99, pp. 826–831, 1914.
109. Millman, T. The use of intoxicating liquors and life insurance. Med. Exam. and Pract., Vol. 15, pp. 94–99, 1905.
110. McMahon, T. F. Alcohol and life insurance. Dominion M. Monthly, Toronto, Vol. 29, pp. 52–59, 1907.
111. Hamilton, C. M. Alcohol and life insurance. Med. Exam. and Pract., N. Y., Vol. 17, pp. 330–334, 1907.
112. Jacquet, L. L'influence de l'alcool sur la mortalité. Monde méd. Paris, T. 22, pp. 392–397, 1912.
113. Neison, F. G. P. On the rate of mortality among persons of intemperate habits. Jour. Stat. Soc. London, Vol. 14, pp. 200–219, 1851.
114. Fernet, C. Motion relative à l'alcoolisme et à la syphilis comme causes de mortalité. Bull. et Mém. Soc. Méd. d. Hop. de Paris, Ser. 3, T. 23, pp. 22–24, 1906.
115. *Id.* De l'alcoolisme et de la syphilis causes de décès dans les hôpitaux et hospices de Paris. *Ibid.*, pp. 1073–78, 1906.
116. *Id.* De la mortalité par alcoolisme et par syphilis dans la hôpitaux et hospices de Paris. *Ibid.*, T. 24, pp. 1016–25, 1907.
117. Pfister, R. Die Trunksucht als Todesursache in Basel in den Jahren 1892–1906. Virchow's Arch. f. path. Anat. Bd. 193, pp. 290–322, 1908.
118. Tamburini, A. Le morti per pellagra, alcoolismo, epilessia e paralisi progressiva in Italia. Riv. sper. di freniat. T. 36, pp. 112–119, 1910.

119. Hatton, E. H. Ratio between deaths from traumatic fracture of cranial bones and from alcohol. Jour. Amer. Med. Assoc., Vol. 77, pp. 2109–2111, 1921.

120. Hindhede, M. Alcohol restriction and mortality. Brit. Med. Jour., Vol. II, pp. 248–252, 1922.

121. McNally, W. D. A comparison of mortalities from alcohol. A report from the coroner's office of Cook County, Illinois. Jour. Amer. Med. Assoc., Vol. 83, pp. 1680–1681, 1924.

122. Stevenson, T. H. C. The mortality of alcoholism. Practitioner, Vol. 113, pp. 270–280, 1924.

123. Vernon, H. M. Alcoholism in various social classes. Lancet, Vol. I, pp. 109–110, 1924.

124. Stewart, H. C., *et al.* Report of a committee of the Harveian Society appointed by the Council in pursuance of a resolution of the Society for the purpose of enquiring into the mortality referable to alcohol. Brit. Med. Jour., 1883 (Vol. I), pp. 97–100.

125. Phelps, E. B. The Mortality of Alcohol: A Statistical Approximation of the Deaths in the United States in which Alcohol may figure as a Causative or Contributory Factor. New York, 8vo, 1911.

126. *Id.* The mortality from alcohol in the United States; the results of a recent investigation of the contributory relation of alcohol with each of the assigned causes of adult mortality. Tr. Internat. Cong. Hyg. and Demog., 1912. Washington, Vol. 1, Part II, pp. 813–829, 1913.

127. Owen, I. Report on the inquiry into the connection of disease with habits of intemperance. Brit. Med. Jour., June 23, 1888, pp. 1309–1320.

128. Fisher, A. Oratorical and statistical fallacies. Year Book of the U. S. Brewers Assoc. 1916, pp. 176–188, 1916.

129. Farr, W. Vital Statistics. (Edited by Noel A. Humphreys.) London, pp. xxiv + 563. 1885.

130. Emminghaus, A. Alkoholismus und Lebensversicherung. Alkoholismus, Bd. 1, pp. 38–44, 1900.

131. Guttstadt, A. Sterblichkeitsverhältnisse der Gastwirte und anderen männlicher Personen in Preussen, welche mit der Erzeugung, dem Vertriebe und dem Verkaufe Alkoholhaltiger Getränke gewerbsmässig beschäftigt sind, in Vergleiche zu anderen wichti-

gen Berufsklassen. Klin. Jahrb., Jena, Bd. 12, pp. 263–294, 1904.

132. Andrae. Die Sterblichkeit in den Berufen die sich mit der Herstellung und dem Verkaufe geistiger Getränke befassen. Mon. Blatt. von Gotha, Nr. 9 and 10, 1905.

133. *Id.* La mortalité d'après les professions, en particulier d'après celles qui ont rapport à la fabrication et à la vente des boissons alcooliques. Ann. d'hyg. 4 S., T. 5, pp. 557–560, 1906. (A digest in French of 132.)

134. Bl., H. Die Sterblichkeit in den Alkoholgewerben nach den Erfahrungen der Gothaer Lebensversicherungsbank von 1852–1902. Internat. Monatschr. z. Erforsch. d. Alkoholismus. Basel, Vol. 15, pp. 193–200, 1905.

135. Medico-Actuarial Mortality Investigation, 1912–1914. New York, Vols. 1–4.

136. Hoppe, H. Mortalität und Morbidität im Brauergewerbe. Alkoholfrage, Bd. 4, pp. 345–353, 1907. (A *Sammelreferat.*)

137. Tatham, J. Letter to the registrar-general, on the mortality of males engaged in certain occupations in its three years 1890–92; and on an English healthy district life table for the ten years 1881–90. London, 1897, pp. cxciv + 166. Part II of Supplement 55, Ann. Rept. Reg. Gen. in England and Wales.

138. Hunter, A. An Address on the Effect of Alcohol on Longevity. Battle Creek, Mich., 8vo, 1914.

139. *Id.* The Medico-Actuarial Investigation of the mortality of American and Canadian life assurance companies. Jour. Inst. Act., Vol. 48, pp. 186–197, 1914.

140. Deuchar, D., Sprague, T. B., and Low, G. M. Mortality in the liquor trade. Investigation by the Associated Scottish Life Offices. *Ibid.* Vol. 33, pp. 245–261, 1897.

141. Holitscher. Die Belastung der Krankenkassen durch die Alkoholiker. Internat. Monatschr. z. Erforsch. d. Alkoholismus, Bd. 20, pp. 233–244; 273–283, 1910.

142. Bertillon, J. On mortality and the causes of death according to occupations. Proc. 15th Internat. Cong. on Hyg. and Demog., Vol. I, pp. 336–369, 1913.

143. Vernon, H. M. Alcoholism in various social classes. Lancet, Vol. I, pp. 109–110, 1924.

144. Neison, F. G. P. The influence of occupation upon health, as

shown by the mortality experienced. Jour. Inst. Act., Vol. 17, pp. 95–126, 1872.

145. Stott, J. On the mortality among innkeepers, publicans, and other persons engaged in the sale of intoxicating liquors — being the experience of the Scottish Amicable Life Insurance Society during fifty years, 1826–1876. *Ibid.* Vol. 20, pp. 35–43, 1876.

146. Neison, F. G. P. The Rates of Mortality and Sickness according to the experience for the ten years 1878–1887 of the Independent Order of Rechabites (Salford Unity) Friendly Society. London, 1889.

147. Advisory Committee of the Central Control Board (Liquor Traffic). Alcohol: Its Action on the Human Organism. London, H. M. Stat. Office, 1918. (This is the first edition of reference 29 in this bibliography.)

148. Porter, W. E. Comparative mortality experience among abstainers and non-abstainers from alcohol. Med. Rec., Vol. 88, pp. 732–737, 1915.

149. Phelps, E. B. The supposed death-rates of abstainers and non-abstainers and their lack of scientific value. American Underwriter Mag. and Ins. Rev., Vol. 40, No. 1, July, 1913.

150. *Id.* Relative death-rates of self-declared abstainers and moderate drinkers from the actuaries' view-point. *Ibid.* June, 1915.

151. Lounsberry, R. L. Mortality experience, total abstainers. Med. Rec. Vol. 84, pp. 986–987, 1913.

152. Nicholl, C. C. British analysis of abstainers mortality. Eastern Underwriter, Dec. 4, 1925, pp. 7–8.

153. Emminghaus, A. Noch einmal Alkolismus und Lebensversicherung. Alkoholismus, Bd. 1, pp. 373–374, 1900.

154. Carpenter, T. M. Human Metabolism with Enemata of Alcohol, Dextrose, and Levulose. Carnegie Institution Publ. No. 369, 1925.

155. Puri, A. N. Effect of methyl and ethyl alcohol on the growth of barley plants. Ann. Bot., Vol. 38, pp. 745–752, 1924.

156. Pearl, R., and Allen, A. The influence of alcohol upon the growth of seedlings. Jour. Gen. Physiol., Vol. 8 (Jacques Loeb Memorial Volume), pp. 215–231, 1926.

157. Southgate, H. W., and Carter, G. Excretion of alcohol in the urine as a guide to alcoholic intoxication. Brit. Med. Jour., March 13, 1926, pp. 463–469.

158. Afanassijew, W. A. Zur Pathologie des akuten und chronischen Alkoholismus. Experimentelle Untersuchung. Ziegler's Beitr. Bd. 8, pp. 443–459, 1890.

159. Rosenfeld, G. Beiträge zur Pathologie des Alkohols. Centralbl. f. inn. Med. Bd. 21, pp. 1049–1060, 1900.

160. Ford, W. W. Obstructive biliary cirrhosis. Amer. Journ. Med. Sci., Vol. 121, pp. 60–85, 1901.

161. Marckwald. Zur Aetiologie und experimentelle Erzeugung der Lebercirrhose. München. Med. Wchnschr. Bd. 48, pp. 489–492, 1901.

162. Crook, J. K. On the etiology of cirrhosis of the liver. Med. News, N. Y., Vol. 80, pp. 246–249, 1902.

163. Dubois, J. Du rôle des vins platrés dans l'étiologie de la cirrhose alcoolique. Lyon (Thesis), 1903. p. 42.

164. Gilbert, A., et Lerebouillet, P. Cholémie familiale et cirrhoses alcooliques. Compt. rend. Soc. de biol. T. 55, pp. 1378–1380, 1903.

165. Boix, E. Référendum sur le rôle de l'alcool, des boissons et produits divèrs dans la production des cirrhoses hepatiques. Arch. gén. de méd. T. 1 and 2, pp. various between 50 and 1575 in T. 1, and 1705 and 2282, in T. 2, 1903.

166. Joannovics, G. Ueber experimentelle Lebercirrhose. Wien. klin. Wchnschr. Bd. 17, pp. 757–762, 1904.

167. Litterer, W. Etiology and pathology of atropic cirrhosis of the liver. Southern Pract. Nashville, Vol. 27, pp. 134–139, 1905.

168. Klopstock, F. Alkoholismus und Lebercirrhose. Virchow's Arch. f. path. Anat. Bd. 184, pp. 304–324, 1906.

169. *Id.* Zur Lehre von der Lebercirrhose. Berliner klin. Wchnschr. Bd. 47, pp. 1532–1535, 1574–1577, 1910.

170. Pearce, R. M. Experimental cirrhosis of the liver. Jour. Exp. Med., Vol. 8, pp. 64–73, 1906.

171. Lancereaux, E. Sur les désordres pathologiques causès par l'abus prolongé du vin. Jour. de méd. de Bordeaux, T. 37, pp. 421–423, 1907.

172. Aubertin, C., et Hébert, P. Hyperhépatie et surcharge glycogenique du foie dans l'intoxication alcoolique expérimentale. Compt. rend. Soc. de Biol., T. 64, pp. 999–1001, 1908.

173. Fischler. Ueber experimentell erzeugte Lebercirrhose. Deutsches Arch. f. Klin. Med. Bd. 93, pp. 427–455, 1908.

174. Meyer, O. Ueber den heutigen Stand der Lehre von der Lebercirrhose. München. Med. Wchnschr. Bd. 55, pp. 2276–2280, 1908.

175. Ribbert, H. Zur Genese der Lebercirrhose. Deutsche med. Wchnschr. Bd. 34, pp. 1678–1681, 1908.

176. Benedict, A. L. The frequency of hepatic sclerosis in non-alcoholics. Med. Rec., Vol. 76, pp. 1075–1076, 1909.

177. Piéry. Les données récentes sur l'étiologie et l'anatomie pathologique des cirrhoses du foie. Lyon méd. T. 113, pp. 997–1014, 1909.

178. Saltykow. Beitrag zur Kenntnis der durch Alkohol hervorgerufenen Organveränderungen. Verhandl. d. Deutsch. path. Gesellsch. Bd. 14, pp. 228–233, 1910.

179. Dal Lago, G. Alcoolismo e cirrosi epatica in Venezia. Riv. veneta di sc. med., T. 55, pp. 145–160, 1911.

180. Rogers, L. Gleanings from the Calcutta post-mortem records. IV. Cirrhosis of the Liver. Indian Med. Gaz., Vol. 46, pp. 47–57, 1911.

181. Bischoff, M. Neue Beiträge zur experimentellen Alkoholforschung, mit besonderer Berücksichtigung der Herz und Leber Veränderungen. Ztschr. f. exper. Path. u. Therap. Bd. 11, pp. 445–466, 1912.

182. Kern, W. Ueber Leberveränderungen bei chronischem Alkoholismus. Ztschr. f. Hyg. Bd. 73, pp. 143–153, 1913.

183. Lissauer, M. Experimentelle Leberzirrhose nach chronischer Alkolholvergiftung. Deutsche med. Wchnschr. Bd. 39. pp. 18–20, 1913.

184. *Id.* Die experimentelle Lebercirrhose. Berl. klin. Wchnschr. Bd. 51, pp. 114–118, 159–163, 1914.

185. *Id.* Leberzirrhose bei experimenteller Intoxikation. Virchow's Arch. f. path. Anat. Bd. 217, pp. 56–62, 1914.

186. Friedenwald, J., and Leitz, T. F. Further experiments on the pathological effect of alcohol on rabbits. Alkoholfrage, N. F. Bd. 10, pp. 124–127, 1913–14.

187. Fox, H. Cirrhosis of the liver in wild animals. New York Med. Jour., Vol. 100, pp. 1209–1213, 1914.

188. Grover, A. L. The question of spontaneous cirrhosis of the liver in rabbits and other laboratory animals. Jour. Amer. Med. Assoc., Vol. 64, pp. 1487–1488, 1915.

189. *Id.* Experimental alcoholic cirrhosis of the liver. Arch. Int. Med., Vol. 17, pp. 193–202, 1916.

190. McJunkin, F. A. The human and animal liver after alcohol. *Ibid.* Vol. 19, pp. 786–800, 1917.

191. Chvostek, F. Zur Pathogenese der Leberzirrhose. Münch. med. Wchnschr. Bd. 65, p. 114, 1918.

192. *Id.* Zur Pathogenese der Lebercirrhose. Ztschr. f. ang. Anat. Bd. 4, pp. 117–146, 1919.

193. Fischer, W. Zur Kenntnis der Leberzirrhose in China. Arch. f. Schiffs- u. Tropen-Hyg. Bd. 23, pp. 435–442, 1919.

194. Ogata, S. Studies on cirrhosis of the liver following intra-portal injection of toxic substances. Jour. Med. Res., Vol. 40, pp. 103–122, 1919.

195. Miller, J. L. The effect of prohibition on the incidence of portal cirrhosis. Jour. Amer. Med. Ass., Vol. 76, pp. 1646–1647, 1921.

196. Villaret, M., Bénard, H., et Blum, P. Contribution à l'étude étiologique des cirrhoses chroniques dites alcooliques. Arch. d. mal. d. l'appar. digest., T. 12, pp. 305–316, 1922.

197. Sergent, É., et Pignot, J. Fréquence actuelle et caractères particuliers de la cirrhose alcoolique du foie chez la femme. Bull. Acad. de Méd. Paris, 3 S. T. 91, pp. 730–733, 1924.

198. Dewar, D. Indian Bird Life, or the Struggle for Existence of Birds in India. London (John Lane), 1925. Pp. xv + 276.

199. Bateson, W. Evolutionary faith and modern doubts. Science, Vol. 55, pp. 55–61, 1922.

200. Richter, C. P. A study of the effect of moderate doses of alcohol on the growth and behavior of the rat. Jour. Exper. Zool., Vol. 44, pp. 397–418, 1926.

201. Phoenix, S. W. The Whitney Family of Connecticut and its Affiliations; being an Attempt to Trace the Descendants, as well in the Female as the Male Lines, of Henry Whitney, from 1649 to 1878; to which is Prefixed Some Accounts of the Whitneys of England. New York (Privately Printed), 1878. 3 vols.

202. Walworth, R. H. Hyde Genealogy; or the Descendants in the Female as well as in the Male lines from William Hyde, of Norwich, with their places of residence, and dates of births, marriages, etc., and other particulars of them and their families and ancestry. Albany (Munsell), 1864. 2 vols.

203. Cope, G. Genealogy of the Smedley family descended from George

and Sarah Smedley, settlers in Chester County, Penna., with brief notices of other families of the name, and abstracts of early English wills. Lancaster, Pa. (Wickensham Printing Co.), 1901. Pp. xi + 1000.

204. Spooner, T. Records of William Spooner, of Plymouth, Mass., and his Descendants. Cincinnati, 1883.

205. Stackpole, E. S., and Meserve, W. S. History of the Town of Durham, New Hampshire (Oyster River Plantation) with Genealogical Notes. Concord, N. H. (Rumford Press), 1913.

206. Shand, P. M. A Book of Wine. London (Guy Chapman), 1926. Pp. xviii + 320.

207. Cabot, R. C. Studies on the action of alcohol in disease, especially upon the circulation. Trans. Assoc. Amer. Physicians, Vol. 18, pp. 402–426, 1903.

208. Lieb, C. C. The reflex effect of alcohol on the circulation. Jour. Amer. Med. Assoc., Vol. 64, pp. 898–903, 1915.

209. *Id.* The reflex effects of alcohol on the circulation. Med. and Surg. Report. Roosevelt Hosp., N. Y., 1915, pp. 199–202.

210. Engelen, P. Die Beeinflussung des systolischen Blutdruckes durch Alkohol. Aertzl. Rundschau. Bd. 31, p. 119, 1921.

211. Dixon, W. E. The action of alcohol on the circulation. Jour. Physiol., Vol. 35, pp. 346–366, 1906–07.

212. Bickel, A. Ueber den Einfluss des Alkohols auf die Herzgrösse. München. med. Wchnschr. 1903 (II), p. 1770.

213. Bingel, A. Untersuchungen über den Einfluss des Biertrinkens und Fechtens auf das Herz junges Leute. *Ibid.* Bd. 54, pp. 57–59, 1907.

214. Mitchell, E. Heart disease, Bright's disease, and alcohol. Trans. New Hampshire Med. Soc., 1901, pp. 181–202.

215. Lian, C. L'alcoolisme, cause d'hypertension artérielle. Bull. Acad. de Méd. Sér. 3, T. 74, pp. 525–528, 1915.

216. Kootataladse, T. G. [The active principles of grape wine.] Russ. Physiol. Jour., Vol. 2, pp. 1–14, 1919.

217. Wrzosek, A. Ueber den Einfluss des Alkohols auf das Wachstum der Mäusecarcinome. Ztschr. f. Krebsforsch. Bd. 11, pp. 515–526, 1912.

218. Tsurumi, M. L'influence de l'alcool éthylique sur le développement des cancers de souris. Ann. d. l'Inst. Pasteur, T. 30, pp. 346–356, 1916.

219. Follet. Réfractaires à l'alcool. Arch. gén. de méd. T. 1, pp. 1544–1546, 1903.

220. Hultgen, J. F. Alcohol and nephritis: A clinical study of 460 cases of chronic alcoholism. Jour. Amer. Med. Assoc., Vol. 55, pp. 279–281, 1910.

221. MacNider, W. deB. A preliminary paper concerning the toxic effect of certain alcoholic beverages for the kidney of normal and naturally nephropathic dogs. Jour. Pharmacol. and Exper. Therap., Vol. 26, pp. 97–104, 1925.

222. *Id.* Chronic nephropathy induced by prolonged administration of an alcoholic beverage. Recovery experiments. Proc. Soc. Exper. Biol., and Med., Vol. 23, pp. 52–56, 1925.

223. Clopatt, A. Ueber die Einwirkung des Alkohols auf den Stoffwechsel des Menschens. Skand. Arch. f. Physiol., Vol. 11, pp. 354–371, 1901.

224. Meyer, E. Ueber den Einfluss der Alkolika auf die sekretorische und motorische Tätigkeit des Magens. Klin. Jahrb. Bd. 13, pp. 285–308, 1905.

225. Kast, L. Experimentelle Beiträge zur Wirkung des Alkohols auf den Magen. Arch. f. Verdauungskrankh. Bd. 12, pp. 487–507, 1906.

226. Haneborg, A. O. The effects of alcohol upon digestion in the stomach. Acta med. Scandin. Bd. 55, Suppl. I, pp. 1–123, 1921.

227. Reich, H. W. Ueber den Einfluss des Alkoholgenusses auf Bakterizidie, Phagozytose, und Resistenz der Erythrozyten beim Menschen. Arch. f. Hyg. Bd. 84, pp. 337–384, 1915.

228. Billings, J. S. Data relating to the use of alcoholic drinks among brain workers in the United States. *In* Physiological Aspects of the Liquor Problem. Boston (Houghton, Mifflin Co.), 1903. 2 vols.

229. Mast, S. O., and Ibara, Y. Effect of ethyl alcohol on tadpoles. Amer. Jour. Physiol., Vol. 59, pp. 294–297, 1922–23.

230. Hecker, R. Ueber Verbreitung und Wirkung des Alkohols bei Schülern. Jahrb. f. Kinderh. Bd. 63, pp. 470–495, 1906.

231. Maignon, F. Sur la présence normal de l'alcool et de l'acétone dans les tissues et liquides de l'organisme. C. R. Ac. Sci., Paris, T. 140, pp. 1063–1065, 1905.

232. Ford, W. H. On the presence of alcohol in normal blood and tissues

and its relation to calorification. Jour. Physiol., Vol. 34, pp. 430–443, 1906.

233. Reach, F. Ueber das Vorkommen von Aethylalkohol und Aethylester im Tierkörper. Biochem. Ztschr. Bd. 3, pp. 326–334, 1907.

234. Schweisheimer, W. Der Alkoholgehalt des Blutes unter verschiedenen Bedingungen. Deutsch. Arch. Klin. Med. Bd. 109, pp. 271–313, 1912–13.

235. Kühn, G. Untersuchungen über Alkohol. II. Mittheilung: Ueber den Alkoholgehalt des menschlichen Blutes im nüchternen Zustand, nach Kohlehydratzufuhr, und nach Genuss geringer Alkoholmengen. Arch. f. exper. Path. u. Pharmakol. Bd. 103, pp. 295–312, 1924.

236. Elderton, E. M., and Pearson, K. A first study of the influence of parental alcoholism on the physique and ability of the offspring. Eugenics Lab. Mem. X, 2nd Edit. pp. 1–46, 1910.

237. Pearson, K., and Elderton, E. M. A second study of the influence of parental alcoholism on the physique and ability of the offspring. *Ibid.* XIII, pp. 1–35, 1910.

238. Barrington, A., and Pearson, K. A preliminary study of extreme alcoholism in adults. *Ibid.* XIV, pp. 1–55, 1910.

239. Stockard, C. R. Alcohol as a selective agent in the improvement of racial stock. Brit. Med. Jour., Aug. 12, 1922, pp. 255–259.

240. *Id.* Experimental modification of the germ-plasm and its bearing on the inheritance of acquired characters. Proc. Amer. Phil. Soc., Vol. 62, pp. 311–325, 1921.

241. Pearl, R. On the effect of continued administration of certain poisons to the domestic fowl, with special reference to the progeny. Proc. Amer. Phil. Soc., Vol. 55, pp. 243–258, 1916.

242. Danforth, C. H. Evidence that germ cells are subject to selection on the basis of their genetic potentialities. Jour. Exp. Zool., Vol. 28, pp. 385–412, 1919.

243. Pictet, A. Résultats négatifs d'expériences d'alcoolisme sur les cobayes. Sur l'apparition de cobayes anormaux dans des lignées non-alcoolisées. Compt. rend. Soc. de phys. et d'hist. nat. de Genève. T. 41, pp. 29–33, 1924.

244. *Id.* Action des vapeurs d'alcool éthylique sur le développement et la pigmentation des Lépidoptères. *Ibid.*, pp. 33–37, 1924.

245. Hanson, F. B. Modification in the albino rat following treatment

with alcohol fumes and X-rays; and the problem of their inheritance. Proc. Amer. Phil. Soc., Vol. 62, pp. 301–310, 1923.

246. Hanson, F. B., and Handy, V. The effects of alcohol fumes on the albino rat. I. Introduction and sterility data from the first treated generation. Amer. Nat., Vol. 57, pp. 532–544, 1923.
247. Hanson, F. B., and Heys, F. Correlation of body weight, body length, and tail length in normal and alcoholic rats. Genetics, Vol. 9, pp. 368–371, 1924.
248. *Id.* Alcohol and the sex ratio. *Ibid.* Vol. 10, pp. 351–358, 1925.
249. MacDowell, E. C., and Vicari, E. M. Alcoholism and the behavior of white rats. I. The influence of alcoholic grandparents upon maze-behavior. Jour. Exper. Zool., Vol. 33, pp. 209–291, 1921.
250. MacDowell, E. C. The influence of alcohol on the fertility of white rats. Genetics, Vol. 7, pp. 117–141, 1922.
251. *Id.* Experiments with alcohol and white rats. Amer. Nat., Vol. 56, pp. 289–311, 1922.
252. *Id.* Alcoholism and the growth of white rats. Genetics, Vol. 7, pp. 427–445, 1922.
253. *Id.* Alcoholism and the behavior of white rats. II. The maze-behavior of treated rats and their offspring. Jour. Exper. Zool., Vol. 37, pp. 417–456, 1923.
254. *Id.* The genetic significance of the alcoholic treatment of white rats. Second Int. Cong. of Eugenics, Vol. I, pp. 168–169, and 428–429 (discussion), 1923.
255. Arlitt, Ada H., and Wells, H. G. The effect of alcohol on the reproductive tissues. Jour. Exper. Med., Vol. 26, pp. 769–778, 1917.
256. Arlitt, A. H. The effect of alcohol on the intelligent behavior of the white rat and its progeny. Psychol. Monog., Vol. 26, No. 4, 1919.
257. Nice, L. B. Comparative studies of the effect of alcohol, nicotine, tobacco smoke and caffeine on white mice. I. Effects on reproduction and growth. Jour. Exper. Zool., Vol. 12, pp. 133–152, 1912.
258. *Id.* Studies on the effects of alcohol, nicotine and caffeine on white mice. II. Effects on activity. *Ibid.* Vol. 13, pp. 123–151, 1913.
259. *Id.* Further observations on the effect of alcohol on white mice. Amer. Nat., Vol. 51, pp. 596–607, 1917.

260. Bluhm, A. Ueber ein Fall experimenteller Verschiebung des Geschlechtsverhältnisses bei Saügetieren. Sitz. Ber. d. Preuss. Akad. d. Wiss. Bd. 34, pp. 549–556, 1921.

261. Gyllenswärd, C. Bidrag till Frågan om Alkoholverkningars Ärftlighet. Stockholm (P. A. Norstedt und Söner), 1923. Pp. 162.

262. Rost, E., and Wolf, G. Zur Frage der Beeinflussung den Nachkommenschaft durch den Alkohol im Tierversuch. Arch. f. Hyg. Bd. 95, pp. 140–153, 1925.

263. Bilski, F. Ueber Blastophthorie durch Alkohol. Mit Versuchen am Frosch. Arch. Entw. Mech. Bd. 47, pp. 627–653, 1921.

264. Mann, Margaret C. A demonstration of the stability of the genes of an inbred stock of *Drosophila melanogaster* under experimental conditions. Jour. Exper. Zool., Vol. 38, pp. 213–244, 1923.

265. Harrison, J. W. H. A preliminary study of the effects of administering ethyl alcohol to the lepidopterous insect *Selenia bilunaria,* with particular reference to the offspring. Jour. Genetics, Vol. 9, pp. 39–52, 1919.

266. Hrdlička, A. The Old Americans. Baltimore (Williams and Wilkins), 1925. Pp. xiii + 438.

267. Hayward, T. E. On life tables: Their construction and practical application. Jour. Roy. Stat. Soc., Vol. 62, pp. 443–483 and 683–702, 1899; and Vol. 63, pp. 625–636, 1900.

268. *Id.* Notes on life tables. *Ibid.* Vol. 65, pp. 354–358, 1902, and 680–684, 1902.

269. Reynolds, E., and Macomber, D. Fertility and Sterility in Human Marriages. Philadelphia (W. B. Saunders Co.), 1924. Pp. 285.

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P36 Alcohol and
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